

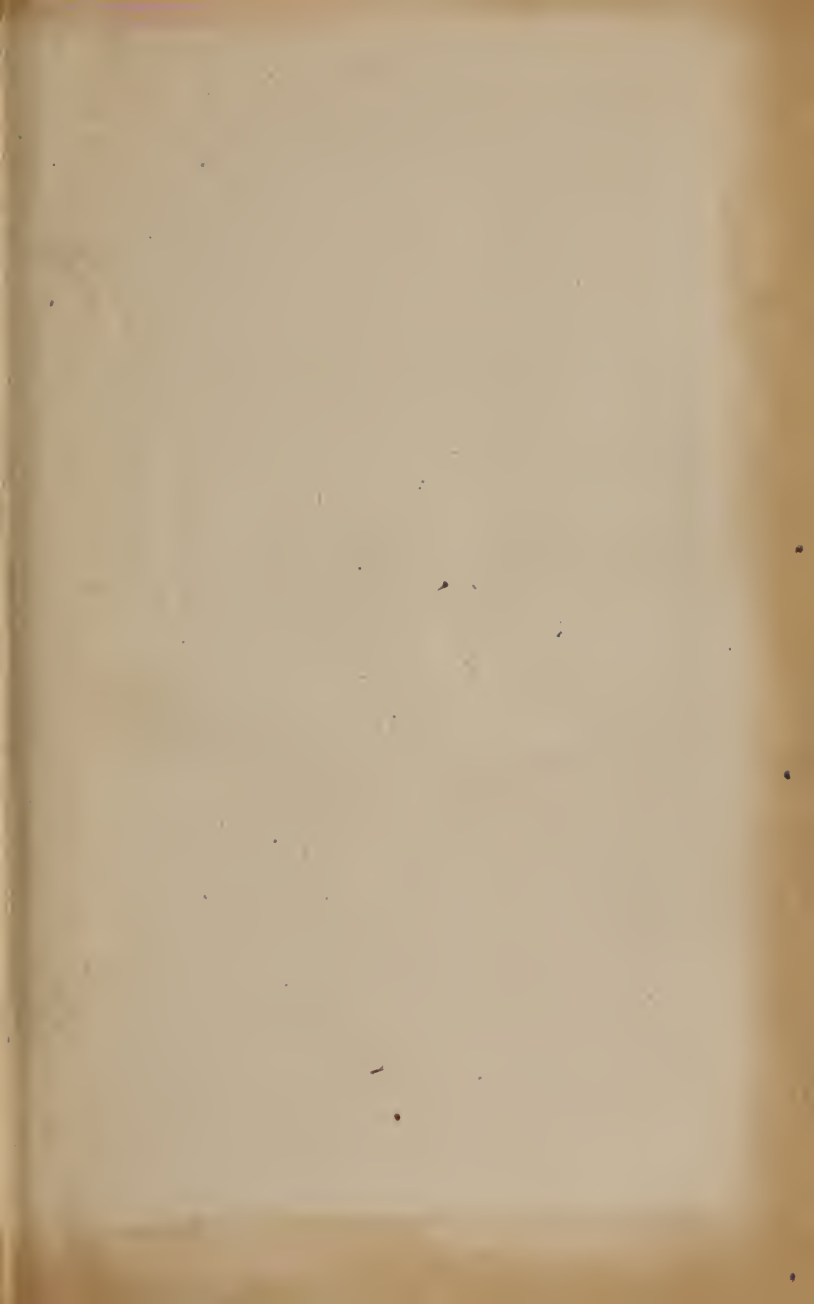
Surgeon General's Office

LIBRARY

~~UNITED~~

Section, *Pathology*

No. *7571* *D*

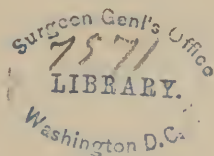


CLINICAL LECTURES
ON
DISEASES OF THE LIVER,

JAUNDICE AND ABDOMINAL DROPSY.

BY
CHARLES MURCHISON, M.D. F.R.S.

Fellow of the Royal College of Physicians; Physician to the Middlesex Hospital;
Lecturer on the Practice of Medicine at the Middlesex Hospital Medical College;
and formerly
On the Medical Staff of H. M.'s Indian Army in Bengal and Burmah.



NEW YORK :
WILLIAM WOOD AND CO., PUBLISHERS,
61 WALKER STREET.
1868.

WIC

M573 c

1868

69.19 no 1

TO
JAMES SYME, ESQ., F.R.S.E.

IN ADMIRATION OF THAT SYSTEM OF

CLINICAL TEACHING

WHICH HAS CONTRIBUTED SO MUCH TO THE RENOWN OF

THE EDINBURGH UNIVERSITY

AS

A SCHOOL OF MEDICINE,

These Lectures are Dedicated

BY

HIS FRIEND AND GRATEFUL PUPIL

THE AUTHOR.

PREFACE.

THESE LECTURES were originally delivered to the students of the Middlesex Hospital, and the first four have already, in part, appeared in the pages of the 'Lancet.' It is hoped that their publication in the present form may be useful not merely to those for whom they were originally intended, but likewise to other members of the Medical Profession.

It is not their object to set forth a complete account of the diseases of which they treat, but rather to put prominently forward the leading characters on which the diagnosis of these diseases mainly depends, and, in particular, to determine the diagnostic import of those signs and symptoms—such as enlargement of the liver, jaundice, dropsy, and pain—which are common to many different hepatic disorders, but the precise cause of which is too often unrecognized.

The original descriptions have in many instances been elucidated by the introduction of diagrammatic representations of physical diagnosis. With the third Lecture has been incorporated a portion of the matter

contained in an essay on 'The Dangers, Diagnosis, and Treatment of Hydatid Tumours of the Liver,' which was published in the 'Edinburgh Medical Journal' for December 1865; and to the last lecture have been added the results of an inquiry into the pathological consequences of gall-stones commenced many years ago, and part of which appeared in a memoir on abdominal fistulæ, published in the 'Edinburgh Medical Journal' for July and August 1857. To all of the lectures has been appended a history not only of those cases on which each lecture was originally founded, but of others which have occurred subsequently and been the subject of clinical remarks in the wards. These histories have been condensed from notes taken at my dictation by my clinical clerks, whose kind and ready assistance I take this opportunity of acknowledging. The records of these cases will, it is believed, be useful to the medical practitioner who meets with others of a like nature.

'Nulla est alia pro certo noscendi via, nisi quamplurimas et morborum et dissectionum historias, tum aliorum, tum proprias, collectas habere, et inter se comparare.'—Morgagni 'De Sed. et Causis Morbor.' Lib. IV. Proœmium.

CONTENTS.



LECTURE I.

ENLARGEMENTS OF THE LIVER.

PAGE

INTRODUCTORY REMARKS—NORMAL DIMENSIONS AND BOUNDARIES OF THE LIVER—CIRCUMSTANCES UNDER WHICH ENLARGEMENT OF THE LIVER IS SIMULATED AND THE MEANS OF DISTINGUISHING SUCH SPURIOUS ENLARGEMENTS: 1. CONGENITAL MALFORMATIONS; 2. EARLY LIFE; 3. RICKETS; 4. TIGHT-LACING; 5. CERTAIN DISEASES OF THE CHEST; 6. TUMOUR BETWEEN THE LIVER AND THE DIAPHRAGM; 7. ABNORMAL CONDITIONS OF THE ABDOMINAL VISCERA; 8. ABNORMAL CONDITIONS OF THE ABDOMINAL PARIETES	1
--	---

LECTURE II.

ENLARGEMENTS OF THE LIVER.

TRUE ENLARGEMENTS OF THE LIVER: SUBDIVISION INTO PAINLESS AND PAINFUL: I. THE WAXY, LARDACEOUS, OR AMYLOID LIVER: II. THE FATTY LIVER; III. SIMPLE HYPERTROPHY . . .	22
--	----

LECTURE III.

ENLARGEMENTS OF THE LIVER.

IV. HYDATID TUMOUR	54
------------------------------	----

LECTURE IV.

ENLARGEMENTS OF THE LIVER.

V. CONGESTION, VI. INFLAMMATION OF THE BILE-DUCTS, VII. OBSTRUCTION OF COMMON DUCT	119
--	-----

LECTURE V.

ENLARGEMENTS OF THE LIVER.

	PAGE
VIII. PYÆMIC ABSCESSES. IX. TROPICAL ABSCESS	147

LECTURE VI.

ENLARGEMENTS OF THE LIVER.

X. CANCER—Rarer Forms of Enlargement: 1. TUBERCULAR.	
2. LYMPHATIC. 3. MULTILOCULAR HYDATIDS. 4. ENLARGEMENTS OF GALL-BLADDER	187

LECTURE VII.

CONTRACTIONS OF THE LIVER.

I. SIMPLE ATROPHY; II. ACUTE OR YELLOW ATROPHY; III. CHRONIC ATROPHY (CIRRHOSIS—SIMPLE INDURATION—RED ATROPHY, &c.)	215
---	-----

LECTURE VIII.

JAUNDICE.

DEFINITION—IMPORTANCE OF RECOGNIZING CAUSES—SPURIOUS JAUNDICE; 1. CHLOROSIS; 2. CANCEROUS CACHEXIA; 3. MALARIA AND POISONS; 4. SUB-CONJUNCTIVAL FAT; 5. ICTERUS NEONATORUM; 6. ADDISON'S DISEASE; 7. EXPOSURE TO SUN; 8. PIGMENTS IN URINE; 9. FEIGNED JAUNDICE. PHENOMENA OF JAUNDICE:—1. LOCALITIES; 2. SECRETIONS; 3. BITTER TASTE; 4. DERANGEMENTS OF DIGESTION; 5. ITCHINESS; 6. CUTANEOUS ERUPTIONS; 7. TEMPERATURE; 8. PULSE; 9. HÆMORRHAGES; 10. GENERAL DEBILITY; 11. YELLOW VISION; 12. CEREBRAL SYMPTOMS—THEORY OF JAUNDICE	279
--	-----

LECTURE IX.

JAUNDICE.

CLASSIFICATION OF CAUSES OF JAUNDICE—JAUNDICE FROM OBSTRUCTION OF THE BILE-DUCT	315
---	-----

LECTURE X.

JAUNDICE.

	PAGE
JAUNDICE INDEPENDENT OF OBSTRUCTION OF THE BILE-DUCT—	
DIAGNOSIS OF THE CAUSES OF JAUNDICE	375

LECTURE XI.

FLUID IN THE PERITONEUM.

ITS SIGNS—THE CONDITIONS WHICH SIMULATE IT, AND HOW TO DISTINGUISH THEM: 1. OVARIAN CYST; 2. HYDATID CYST; 3. RENAL CYST; 4. DISTENDED URINARY BLADDER; 5. PREGNANT UTERUS—CAUSES OF FLUID IN PERITONEUM: I. ACUTE PERITONITIS; II. CHRONIC PERITONITIS; III. CANCER; IV. COLLOID; V. SIMPLE DROPSY—1. FROM DISEASE OF KIDNEYS; 2. FROM DISEASE OF HEART OR LUNGS; 3. FROM PORTAL OBSTRUCTION	432
---	-----

LECTURE XII.

A. HEPATIC PAIN.

SIMULATED BY: 1. PLEURODYNIA; 2. INTERCOSTAL NEURALGIA; 3. PLEURISY; 4. GASTRIC DYSPEPSIA; 5. INTESTINAL COLIC; 6. RENAL COLIC—THE VARIETIES AND CAUSES OF GENUINE HEPATIC PAIN	491
---	-----

B. GALL-STONES.

THEIR VARIOUS CONSEQUENCES, SYMPTOMS, AND TREATMENT	499
---	-----

C. ENLARGEMENTS OF GALL-BLADDER.

THEIR CAUSES, CLINICAL CHARACTERS, AND TREATMENT	536
--	-----

INDEX	547
-----------------	-----

Errata.

Pages 75 to 92, and page 179, line 27, *for* canula *read* cannula.

„ 185, line 15, *for* liver *read* lung.

„ 216, line 9, *for* autero-posterior, *read* antero-posterior.

LIST OF WOOD-ENGRAVINGS.

FIG.	PAGE
1. Natural position of the Liver, as seen after removal of the Anterior Wall of the Chest and Abdomen	3
2. Natural position of the Liver, as seen after the removal of the Vertebrae and the Posterior Wall of the Chest and Abdomen	4
3. Normal area of Hepatic Dulness, viewed anteriorly	5
4. Normal area of Hepatic Dulness, viewed from right side	6
5. Normal area of Hepatic Dulness, viewed posteriorly	6
6. Apparent enlargement of the Liver resulting from Tight-lacing	12
7. Area of dulness caused by effusion into the Right Pleura, depressing the Liver	14
8. Displacement of the Liver downwards by extensive effusion into the Pericardium	15
9. Increased area of Hepatic and of Splenic Dulness from Waxy Disease: anterior view	25
10. Increased area of Hepatic Dulness from Waxy Disease	25
11. Area of Hepatic Dulness in a case of Hydatid Tumour of the Liver	84
12. Area of Hepatic Dulness in a case of Hydatid Tumour of the Liver	86
13. Area of Hepatic Dulness in a case of Enlargement of the Liver, and Distension of the Gall-Bladder from obstruction of the Common Bile-duct .	145

FIG.	PAGE
14. Area of Hepatic Dulness in a case of Tropical Abscess of the Liver	181
15. Area of Hepatic Dulness in a case of Cancer of the Liver	189
16. Microscopic appearances in a case of Fungating Cancerous Tumour of the Liver, showing transitional forms between the Glandular Epithelium and 'Cancer Cells'	205
17. Area of Hepatic Dulness in a case of Acute Atrophy of the Liver	226
18. Microscopic needle-shaped Crystals of Tyrosine adhering in bundles and in stellate groups	230
19. Microscopic globular Masses composed of acicular crystals of Tyrosine	230
20. Microscopic, laminated, crystalline masses of Leucine	231
21. Shows the Hepatic and Ascitic Dulness in a case of Cirrhosis of the Liver	245
22. Microscopic crystalline masses of Carbonate of Lime from the Gall-Bladder	277
23. Microscopic appearances of the Blood in a case of Chronic Atrophy of the Liver with Leukæmia	277
24. Percussion-sounds over the Abdomen in a case of Ascites from Cirrhosis of the Liver	436
25. Percussion-sounds over the Abdomen in a case of Tumour of the Left Ovary	437

LIST OF CASES.

CASE	PAGE
I. Caries of the Hip-joint—Waxy Liver weighing nearly one-seventh of the entire body—Waxy Spleen—Fatty Kidneys	35
II. Constitutional Syphilis, followed by Symptoms of Waxy Disease of the Liver, Spleen, and Kidneys .	37
III. Syphilitic Necrosis of Lower Jaw—Albuminuria—Pleurisy and Pericarditis—Waxy Liver and Kidneys	41
IV. Waxy Liver enlarged and nodulated, simulating Cancer	42
V. Acute Phthisis—Fatty Liver	52
VI. Hydatid Tumour of the Liver—Paracentesis—Recovery	83
VII. Hydatid Tumour of the Liver threatening to burst—Paracentesis—Recovery	85
VIII. Hydatid Tumour of the Liver—Paracentesis—Recovery	89
IX. Hydatid Tumour of Liver opening into the common Bile-duct—Jaundice and Suppuration of the Cyst—Puncture with a large Trocar, and permanent opening—Pneumonia—Death	90
X. Hydatid Tumour of the Liver bursting into the Bile-duct—Jaundice—Discharge of innumerable Hydatid Cysts per anum—Recovery—Attacks of Biliary Colic from passage of Cysts remaining in the Liver through the Bile-duct—Rupture of old Adhesions during the act of Vomiting—Peritonitis—Death	94

CASE	PAGE
XI. Hydatid Cyst of Liver—Entrance of Bile—Inflammation—Paracentesis—Death	97
XII. Suppurating Hydatid Tumour of Liver—Pyæmia with Secondary Deposits of Pus	99
XIII. Suppurating Hydatid—Pyæmia, with Secondary Gangrenous Abscesses in the Liver	100
XIV. Enormous Hydatid Cyst of the Liver, passing down through the Foramen of Winslow, and filling almost the whole of the Abdominal Cavity—Paracentesis—Pleurisy—Tubercle of Lungs—Death from Exhaustion	102
XV. Hydatid Tumour of the Liver, bursting into the Right Pleura—Empyema—Death	104
XVI. Hydatid Tumour of the Liver, opening into the Right Pleura—Empyema—Pericarditis—Death	105
XVII. Old Hydatid Tumour of the Liver, communicating with Base of Right Lung—Lobular Pneumonia and Gangrene of the Lung	106
XVIII. Hydatid Tumour of the Liver—Pyelitis—Pus in the Urine—Sudden Death	107
XIX. Hydatid Tumours of the Liver and Peritoneum compressing the Ureters, and causing Degeneration of the Kidneys	109
XX. Hydatid Cysts of the Liver and Peritoneum—Ascites and Anasarca of lower Extremities—Albuminuria—Death	110
XXI. Hydatid Tumour of the Liver—Secondary Hydatid Tumours in the Spinal Canal—Paraplegia	111
XXII. Large Hydatid Tumour of the Liver full of Collapsed Secondary Cysts, but containing no fluid	112
XXIII. Enormous Cystic Tumour communicating with the Pelvis of the Right Kidney, existing for eight years, and simulating Hydatid Tumour of the Liver	115
XXIV. Mitral Constriction—Dropsy and Congestion of the Liver—Death	128
XXV. Indigestion from habitual Surfeit—Residence in Tropics—Exposure to Chill—Congestion of Liver	130

CASE	PAGE
XXVI. Congestion of the Liver	131
XXVII. Painful Enlargement of the Liver, with Jaundice, due to Inflammation of the Bile-ducts .	135
XXVIII. Gouty Dyspepsia—Jaundice and Enlargement of the Liver from Inflammation of the Bile-ducts	136
XXIX. Inflammation of the Biliary Passages, excited by Gall-stones—Gangrene of Foot—Diseased Kidneys—Death by Uræmia	137
XXX. General Tuberculosis — Enlargement of the Liver from Tubercular Deposit—Jaundice from Inflammation of the Bile-ducts—Embolism of the Spleen	140
XXXI. Enlargement of Liver and Dilatation of Gall-bladder from Obstruction of Common Duct by a Calculus	145
XXXII. Injury of Cranium, followed by Pyæmia and multiple Abscesses in the Liver	154
XXXIII. Multiple Abscesses in the Liver Secondary to Simple Ulcer of the Stomach	155
XXXIV. Multiple Abscesses in the Liver secondary to Cancerous Ulcer of the Stomach	157
XXXV. Attacks of Biliary Colic followed by Pyæmic Abscesses in the Liver	158
XXXVI. Multiple Abscesses of the Liver—Softening Tubercle in Mediastinal Glands	160
XXXVII. Tropical Abscess of the Liver—No dysenteric Ulceration of the Bowel	180
XXXVIII. Large Abscess of the Liver opening into the Ascending Colon	183
XXXIX. Abscess of the Liver opening upwards through Diaphragm—Secondary Abscess of Lung .	185
XL. Tropical Abscess of the Liver—Puncture with a large Trocar—Recovery	186
XLI. Cancer of the Liver and Ovary—Jaundice, but no Ascites	196
XLII. Cancer of Uterus and Liver—Ascites, but no Jaundice	198
XLIII. Cancer of the Liver, Lungs, and Cervical Glands—Jaundice and Ascites	200

CASE	PAGE
XLIV. Primary Cancer of the Liver—Death from Hæmorrhage into the Peritoneum	202
XLV. Cancerous Tumour (Fungus Hæmatodes), projecting from upper surface of Liver—Hæmorrhage into Peritoneum	204
XLVI. Cancer of Vertebrae, Supra-renal Capsule, Liver and Lung—No Symptoms of Disease of Liver	206
XLVII. Melanotic Cancer of the Penis, Lymphatic Glands, Liver, Pleura, &c.	208
XLVIII. Coexistence of Cancerous Stricture of the Œsophagus, with recent Tubercle in the Lungs—Simple Atrophy of the Liver	222
XLIX. Acute Atrophy of the Liver—Acute Peritonitis—Leucine and Tyrosine in the Liver and Kidneys, but none detected in the Urine	236
L. History of Spirit-drinking and Symptoms of portal Obstruction—Dense, fibrous, granular Liver—True Cirrhosis	260
LI. History of Spirit-drinking—Cirrhosis of the Liver—Nephritis—Epileptiform Convulsions and Death by Uræmia	263
LII. History of Spirit-drinking—Contracted Liver—Copious Hæmatemesis—Delirium Tremens	266
LIII. Chronic Atrophy of the Liver—Ascites—Hæmatemesis and Bloody Stools	267
LIV. No history of Spirit-drinking—Symptoms of portal Obstruction—Soft, atrophied, granular Liver—Spurious Cirrhosis	271
LV. Chronic Atrophy of the Liver, from Peri-hepatitis—Simple Ulcers of the Stomach	273
LVI. Bronchitis and Dilated Bronchi—Disease of the Aortic Valves—Contracted Liver—Great Ascites	274
LVII. Chronic Atrophy of the Liver and Ascites—Paracentesis—No Accumulation after fourth Tapping—Enlarged Spleen and Leukæmia—Death from Ulceration of the Mouth and Pharynx, and Necrosis of the Jaw and Vertebrae	275
LVIII. Jaundice from Obstruction of the Bile-duct by . Gall-stones	360

CASE	PAGE
LIX. Jaundice from temporary Obstruction of the Bile-duct by Gall-stones	361
LX. Jaundice from Congenital Closure of the Bile-duct	363
LXI. Jaundice from Obstruction of the Common Bile-duct by the Cicatrix of a Duodenal Ulcer—Dilatation of the Bile-duct and Atrophy of the Liver	365
LXII. Jaundice from Obstruction of the common Bile-duct by a Cancerous Growth from its lining Membrane—Dilatation of the Bile-ducts and Enlargement of the Gall-bladder—Cancerous Growth in the Pancreas	367
LXIII. Cancer of the Pancreas and of the Gall-bladder—Jaundice from Obstruction of the Bile-duct	369
LXIV. Permanent Jaundice from Closure of the Bile-duct	371
LXV. Typhus Fever complicated with Jaundice—Death by Coma—Leucine and Tyrosine, but scarcely any Urea, in Urine—Leucine and Tyrosine in Liver and Kidneys	408
LXVI. Typhus Fever complicated with Jaundice	409
LXVII. Typhus Fever — Double Pleuro-pneumonia — Jaundice—Tyrosine in Urine	410
LXVIII. Typhus Fever followed by Phlegmasia Dolens, Jaundice, and Death	411
LXIX. Enteric Fever followed by a Relapse with Jaundice	413
LXX. Enteric Fever complicated with Jaundice	414
LXXI. Enteric Fever complicated with Jaundice and 'Phlebitis'	415
LXXII. Scarlatina—Jaundice—Death by Coma	415
LXXIII. Scarlatina—Jaundice—Sudden Death	416
LXXIV. Scarlatina—Jaundice—Recovery	417
LXXV. Scarlatina—Jaundice—Recovery	418
LXXVI. Acute Necrosis of Cervical Vertebrae—Pyæmia—Jaundice	418
LXXVII. Acute Pleuro-pneumonia complicated with Jaundice	420
LXXVIII. Jaundice from Congestion of the Liver	421

CASE	PAGE
LXXXIX. Cystic Tumour of the Ovary opening into the Rectum—Entrance of Air into the Ovarian Cyst—Atrophy of Right Lobe of Liver and complementary Hypertrophy of Left Lobe	466
LXXX. Enlargement of the Abdomen from a distended Urinary Bladder mistaken for an Hydatid Tumour of the Liver—480 ounces of Urine drawn off by Paracentesis Abdominis	468
LXXXI. Fluid in Peritoneum from Acute Peritonitis due to a Kick over a Congenital Hernia	471
LXXXII. Fluid in Peritoneum from Chronic Peritonitis—Chronic Atrophy of Liver with Fibroid Nodules in its interior, apparently independent of Syphilis	473
LXXXIII. Primary Cancer of the Peritoneum, causing a large Effusion of Fluid	475
LXXXIV. Tubercular Peritonitis—Signs of a circumscribed collection of Fluid in Peritoneum	477
LXXXV. Symptoms of Colic followed by signs of Fluid in the Peritoneum	480
LXXXVI. Fluid in the Peritoneum from Disease of the Kidney—Albuminuria and General Anasarca—Pericarditis and Pleurisy—Death by Uræmia—Great Hypertrophy of Left Kidney and Atrophy of Right	481
LXXXVII. History of Spirit-Drinking—Cirrhosis of Liver—Enlarged Spleen—Ascites—Gastro-enteritis—Removal of Ascites by Diuretics	484
LXXXVIII. Constriction of Mitral Valve—Chronic Atrophy of Liver—Ascites and Jaundice	486
LXXXIX. Great Ascites and Diarrhœa—Enlargement of the Spleen and Liver and Albuminuria	488
XC. Hepatic Neuralgia	497
XC1. Gall-stones in a Sacculus of the common Bile-duct and in the Gall-bladder—Ulceration and Perforation of the Gall-bladder—Fatal Peritonitis	527

CASE	PAGE
XCII. Fistulous Opening between the Gall-bladder and the Duodenum—Fatal Obstruction of the Small Intestine by a large Biliary Calculus . . .	529
XCIII. Closure of the Cystic Duct—Abscess of the Gall-bladder—Discharge of Gall-stones through a Fistulous Opening in the Abdominal Parietes .	531
XCIV. Fistula in the Abdominal Parietes, opening into a circumscribed Cavity which communicated with the Colon and the Duodenum, and also with the Gall-Bladder	534
XC.V. Cancer of the Rectum, Secondary Cancer of the Liver and Gall-bladder obliterating the Cystic Duct—Enlargement of the Gall-bladder . . .	543
XCVI. Destruction by Cancerous Ulceration of the Gall-bladder, and communication of the resulting Cavity with the Transverse Colon—Cancer of the Liver.	543

CLINICAL LECTURES

ON

DISEASES OF THE LIVER AND JAUNDICE.

LECTURE I.

ENLARGEMENTS OF THE LIVER.

INTRODUCTORY REMARKS—NORMAL DIMENSIONS AND BOUNDARIES OF THE LIVER—CIRCUMSTANCES UNDER WHICH ENLARGEMENT OF THE LIVER IS SIMULATED, AND THE MEANS OF DISTINGUISHING SUCH SPURIOUS ENLARGEMENTS: 1. CONGENITAL MALFORMATIONS; 2. EARLY LIFE; 3. RICKETS; 4. TIGHT-LACING; 5. CERTAIN DISEASES OF THE CHEST; 6. TUMOUR BETWEEN THE LIVER AND DIAPHRAGM; 7. ABNORMAL CONDITIONS OF THE ABDOMINAL VISCERA; 8. ABNORMAL CONDITIONS OF THE ABDOMINAL PARIETES.

GENTLEMEN,—In systematic lectures on Medicine, it is the custom to describe in detail the numerous symptoms which characterise different disorders. It requires, however, little experience to discover that there are symptoms and signs which are common to many diseases, and that no small difficulty is often encountered in determining to which of its many sources a particular symptom ought to be referred. Yet this determination must always be your first object in practice. You must never rest satisfied with treating merely a symptom without endeavouring to acquire some definite notion of the local or general

disease on which it depends. In all cases of disease presenting some prominent symptom, you ought to ask yourselves two questions: 1. What are the different causes which may give rise to the symptom in question? and 2. Which is the most probable cause in the individual case before you? Not until you have given a satisfactory reply to these enquiries will you be in a position to speak with any confidence as to prognosis, or to adopt a rational method of treatment.

To no class of maladies are these remarks more applicable than to diseases of the liver. There are few diseases more difficult to discriminate, and perhaps none in which an erroneous diagnosis is oftener made; while symptoms depending upon disease of the stomach, the intestines, or the kidneys, or even the heart, the lungs, or the brain, are constantly ascribed to derangements of the liver. It will be my object in these lectures to point out to you the chief signs and symptoms resulting from hepatic disease, the different morbid conditions from which each of them may arise, the rules by which you must be mainly guided in determining the precise disease in each case, and the conclusions to which you ought in this way to be led respecting prognosis and treatment. We shall commence, for instance, by discussing the different causes of Enlargement of the Liver; and in subsequent lectures, the causes of Atrophy of the Liver, of Jaundice, Hepatic Pain, Hepatic Dropsy, &c., will be duly considered.

ENLARGEMENTS OF THE LIVER.

Before proceeding to consider the various causes of true enlargement of the liver, it is necessary to have an accurate knowledge of its normal dimensions and boundaries, and also to keep in view certain conditions which during life may simulate enlargement.

Normal situation and dimensions of the liver.—The liver is situated in the right hypochondrium, the convexity of the right lobe corresponding to the concavity at the base of the right lung with the dia-

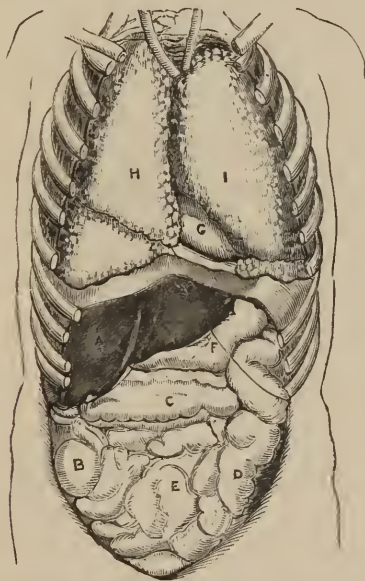


Fig. 1. Natural Position of the Liver, as seen after removal of the anterior wall of the chest and abdomen. Modified from Sibson's Med. Anat.

A, Liver. B, Ascending colon. C, Transverse colon. D, Descending colon. E, Small intestines. F, Stomach. G, Heart. H, Right lung. I, Left lung.

phragm interposed, and the under surface being opposed to the stomach and large intestine, the right

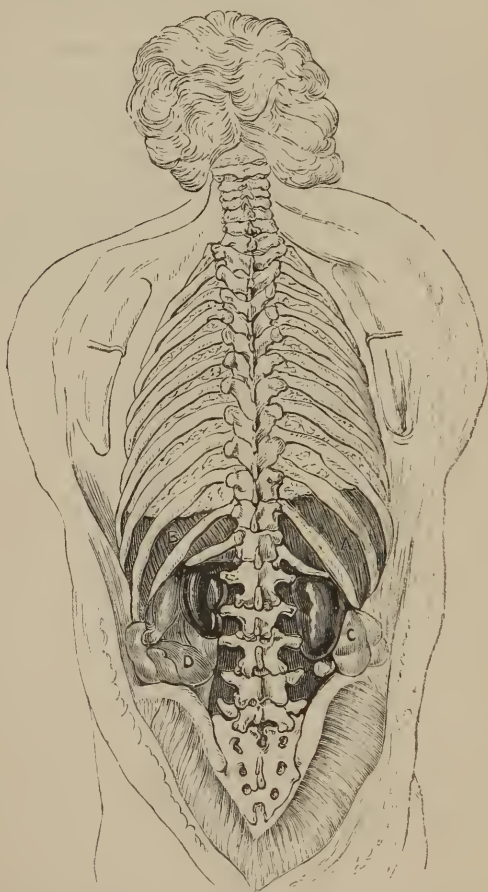


Fig. 2. Natural Position of the Liver, as seen after the removal of the vertebræ and the posterior wall of the chest and abdomen. Modified from Sibson's Med. Anatomy.

The liver is covered by the diaphragm, beneath which, on the left side (B) there is also the spleen and a portion of the stomach. A, Right lobe of liver. C, Ascending colon. D, Descending colon.

kidney and supra-renal capsule. The convex upper surface projects up into the right side of the chest, and a great part of it is in immediate juxtaposition with the ribs, but the uppermost portion (in a vertical direction) is separated from the wall of the chest by the thin lower margin of the right lung. (See fig. 1.) Accordingly, in percussion during life, the upper

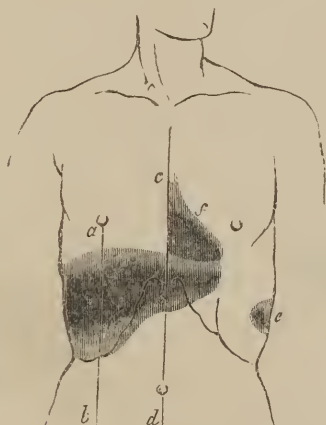


Fig. 3. Area of Hepatic Dulness, viewed anteriorly.

a-b, Right mammary line. *c-d*, Median line. *e*, Splenic dulness. *f*, Cardiac dulness.

margin of hepatic dulness may be said to be twofold, one boundary limiting the region where the organ is in close approximation to the walls of the chest, and where the dulness is absolute, the other corresponding to the extreme height of the liver, and including the space where it is overlapped by the thin layer of lung, and where the sound on percussion constitutes

a transition from the hepatic dulness to the pulmonary resonance. It is the latter which is usually regarded as the true upper margin of the liver (fig. 3).

There is a peculiarity in the upper margin of hepatic dulness which is of some practical importance



Fig. 4. Area of Hepatic Dulness, viewed from right side.

a-b, Right axillary line.



Fig. 5. Area of Hepatic Dulness, viewed posteriorly.

a-b, Right dorsal line. *c*, Splenic dulness. *d*, Left kidney. *e*, Right kidney. *f*, Descending colon. *g*, Ascending colon.

—namely, that it is not horizontal, but arched. Commencing posteriorly about the tenth or eleventh dorsal vertebra, it ascends gradually towards the axilla and the nipple, and then again descends slightly

towards the median line in front. The arched character of the upper surface of the liver is shown in the annexed diagrams (figs. 3, 4, 5).

In determining the upper margin of hepatic dulness we must trust to percussion alone. In ordinary cases it is sufficient to note the upper limit in what is called the *right mammary line*, or a line descending perpendicularly from the right nipple (fig. 3). Here, in a healthy adult, the *true* upper margin of the liver is situated in the fifth intercostal space, or in rare cases behind the fifth rib or in the fourth space. In this line, the liver is overlapped by lung to the extent of about one inch. But in all cases of suspected hepatic disease, the upper margin of hepatic dulness ought to be determined in its entire course. In the *median line* in front, it usually corresponds to the base of the ensiform cartilage, or rises slightly above this.

To the left of the median line it is difficult or impossible to define the upper limit of hepatic dulness from the lower boundary of the heart, the two being in apposition, but a line drawn from the upper margin of hepatic dulness in the median line to the apex of the heart will usually correspond to the line of separation. In the *right axillary line* (fig. 4), or a line falling perpendicularly from the centre of the axilla, the upper margin of hepatic dulness corresponds to the seventh intercostal space, or more rarely, to the seventh rib. In the *right dorsal line*, or a line falling perpendicularly from the lower angle

of the scapula (when the arm is dependent), it corresponds to the ninth intercostal space, or the ninth rib (fig. 5).

The lower margin of hepatic dulness may be determined by percussion, and often also by means of palpation. It is, however, as a rule, less easily defined than the upper margin, being often obscured by a distended condition of the stomach or intestines. Hence it is always most satisfactorily examined when the stomach is empty, and after the bowels have been freely moved. In the right mammary line, it usually corresponds with the margin of the costal arch, or is half an inch above or below this; in the right axillary line, it corresponds to the tenth intercostal space; and in the right dorsal line, to the twelfth rib, although here it is usually difficult to define it from the dulness of the kidney. In the epigastrium, the lower margin of the right and left lobes usually descends two or three inches below the angle of junction of the last costal cartilage with the sternum.

The ordinary extent of hepatic dulness, in an adult of average size, is 4 inches in the right mammary line, $4\frac{1}{2}$ or 5 inches in the right axillary line, 4 inches in the right dorsal line, and 3 or 4 inches in the median line anteriorly.

But it must not be forgotten that, even in the same individual, the liver is constantly liable to slight alterations in its position consistently with health. During the act of inspiration the whole organ is

slightly lowered—about half an inch—and its upper surface is somewhat flattened, whereas during expiration the organ ascends. Again, in the erect position, the lower margin will extend somewhat lower than when the patient is recumbent. If in the mammary line it correspond to the lower margin of the costal arch in the latter position, it may be a quarter of an inch lower in the former. These variations however are slight, and are not likely to embarrass the diagnosis.

But difficulties in diagnosis may sometimes arise from the boundaries of the liver, as above defined, being greatly exceeded without any real enlargement of the organ. In all cases of suspected enlargement of the liver it is important to keep in view the possibility of its being of this spurious character.

CIRCUMSTANCES UNDER WHICH ENLARGEMENT OF
THE LIVER IS SIMULATED DURING LIFE.

The chief of these conditions are the following:—

I. *Congenital malformations, &c.*—In rare cases, in consequence of congenital malformation, the liver is more square or globular than natural, and a larger portion of it is in apposition to the abdominal and thoracic wall. In other cases the left lobe is proportionately large, as in the fœtus. In cases of still greater rarity the liver is protruded into the right side of the chest through an opening in the diaphragm, which may be congenital, or the result of accident. Not long ago a case of this sort came

under my notice, where the greater portion of the right lobe of the liver was lodged in the right pleura, and where the hepatic dulness in consequence ascended as high as the third rib. The particulars of the case will be found in the last volume of the Pathological Society's 'Transactions,' vol. xvii. p. 164. The diagnosis of such conditions during life must of course always be difficult, and will rest mainly on the following conditions:—

1. The absence of any symptom indicative of disease of the liver.

2. The absence of other circumstances likely to produce spurious enlargement.

3. The fact of the increased hepatic dulness persisting from early life (except in diaphragmatic hernia resulting from accident).

II. *Early life.*—The liver is proportionally much larger in infancy and adolescence than in adult life. The organ does not grow in proportion to the rest of the body. In the adult the average weight of the liver is one-fortieth of that of the entire body, whereas previous to puberty it may be as much as one-thirtieth, or even one-twentieth. The dimensions vary accordingly, so that the upper margin of hepatic dulness is often higher in the child than in the adult, and the lower margin descends below the costal arch in the right mammary line. It follows, therefore, that an extent of hepatic dulness which in the adult would be abnormal, may be perfectly normal in the child. In the wards of the hospital I

have had frequent opportunities of pointing out to you this peculiarity of the liver in early life.

III. *Rickets*, causing lateral distortion of the spine, and the deformity known as the 'pigeon breast,' may lead to apparent enlargement of the liver, owing to the organ being depressed and elongated in its vertical diameter from lateral compression. The resemblance to hepatic enlargement may be further increased by there being a disproportionate recession of the ribs immediately above the margin of the liver, as the result of which there is an apparent bulging of the hepatic region. Hence, in lateral distortion of the spine and in the 'pigeon breast,' care must be taken not to arrive at any hasty conclusion as to enlargement of the liver.

IV. *The practice of tight-lacing* may cause displacements and malformations of the liver, which may simulate enlargement, and which are of considerable importance in diagnosis. Tight-lacing may act on the liver in three ways, according to the situation, the tightness, and the duration of the constricting cause.

a. The liver may be displaced upwards or downwards, according as the pressure is applied below or above. The precise situation where the pressure is applied will vary with the prevailing fashion of dress; but most commonly in this country the displacement is downwards, and this may be to such an extent that the lower margin reaches the ilium, and the liver appears to fill up the whole of the right side and front of the abdomen (fig. 6).

b. In consequence of lateral compression, the liver may be elongated in its vertical diameter, so that a larger portion of it is brought into apposition with

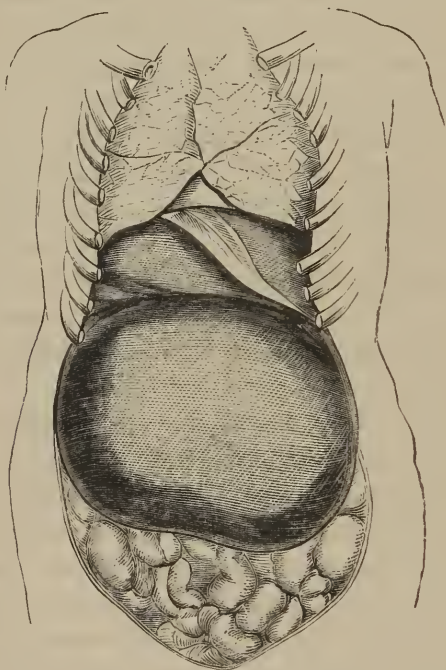


Fig. 6. Apparent enlargement of the Liver resulting from Tight-lacing. Modified from Frerichs. The Liver is depressed, and its vertical diameter elongated. A deep transverse furrow corresponds to the site of constriction.

the abdominal and thoracic walls. This is a very common result of tight-lacing (fig. 6).

c. When the pressure is exerted by a tight cord, it may produce deep fissures in the substance of the

liver, as the result of which portions of the organ may be more or less detached, and may even be felt as movable tumours through the abdominal parietes.

Apparent enlargements of the liver from tight-lacing are far more common than is generally believed. You cannot pay many visits to the post-mortem room without observing examples of this malformation. Moreover, these acquired malformations of the liver, although most common in females, are occasionally observed in the male sex. I show you here the liver of a man with a deep furrow, from indentation of the ribs, which resulted apparently from the practice of wearing a very tight belt. I may also call your attention to the case of a man, aged 23, lately under your observation in the hospital, with a firm movable tumour in the epigastrium, which there was every reason to believe was a portion of the liver partially detached from a similar cause.

Apparent enlargements of the liver from tight-lacing may usually be recognised by the following characters :—

1. Evident signs of tight-lacing in the walls of the chest and abdomen.

2. Occasionally the existence of a distinct transverse furrow in the substance of the liver, appreciable through the abdominal parietes on palpation.

3. The absence of symptoms of structural disease of the liver itself, or of serious disease in the chest or abdomen.

V. *Certain diseases in the chest may cause great*

depression of the liver into the abdominal cavity, and lead to the idea that the organ is enlarged. This remark applies particularly to extensive effusion into the right pleural cavity, or to pneumo-thorax on the right side. In these affections the natural convexity of the diaphragm may be reversed, and the

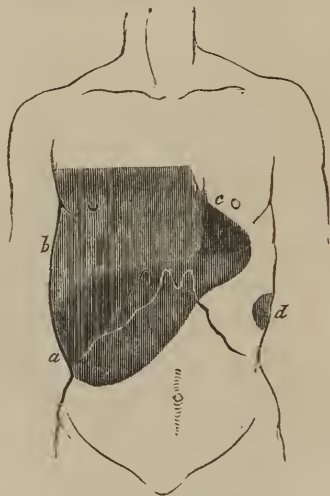


Fig. 7. Effusion into the Right Pleura depressing the Liver.

a, Hepatic dullness. *b*, Dulness from pleuritic effusion causing bulging of the right side of chest, and displacing the heart to the left; its upper margin horizontal. *c*, Cardiac dullness. *d*, Splenic dullness.

lower margin of the liver may descend to the umbilicus (fig. 7). Depression to a less extent may result from intra-thoracic tumours, effusion into the left pleura or into the pericardium (fig. 8); and even in pulmonary emphysema and acute pneumonia* the

* See a case of acute pneumonia of the right lung, referred to by Dr. Stokes in his work on 'Diseases of the Heart and Aorta,' p. 453.

liver may be lowered to the extent of an inch or more. In all cases, however, where the liver is depressed

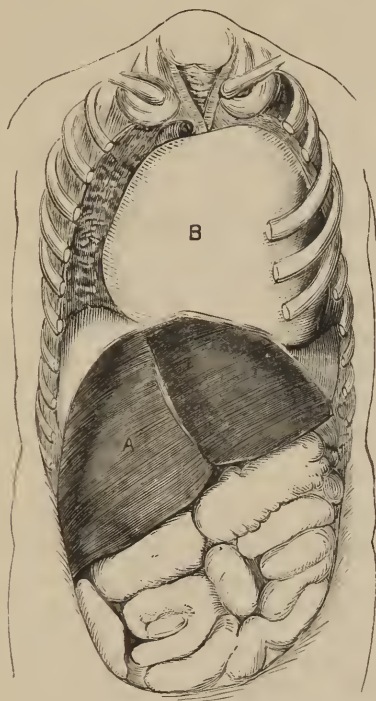


Fig. 8. Displacement of the Liver downwards by extensive Effusion into the Pericardium : after Sibson.

A, Liver. B, Pericardium greatly distended with fluid.

‘So great was the enlargement of the lung that the diaphragm and liver were pushed far down into the abdominal cavity.’ Dr. Bright speaks of displacement of the liver downwards by pneumonic consolidation as a frequent occurrence (*Abdom. Tumours*, Syd. Soc. ed., p. 255) ; but Stokes regards it as exceptional, and this also is the result of my own observation. In extensive pneumonia, however, the liver is usually more or less congested, and enlarged accordingly.

in consequence of disease in the chest, the descent of its lower margin is probably due to a combination of causes; for when there is disease in the chest sufficient to depress the diaphragm, there is usually also congestion with slight enlargement of the liver.

Apparent enlargements of the liver from the causes now referred to have the following distinguishing features:—

1. A previous history of pleurisy, pericarditis, bronchitis and emphysema, pneumonia, or phthisis ending in pneumothorax. At the same time it is well to remember that extensive effusion sometimes takes place into the pleura in a very latent manner.

2. A degree of dyspnœa far greater than would be accounted for by the amount of enlargement of the liver, even if real.

3. The physical signs of the various thoracic diseases above referred to. In the case of emphysema and pneumothorax, there is no difficulty in defining the upper margin of the liver, and in ascertaining that the extent of hepatic dulness is not increased, so that percussion will at once reveal the nature of the case. But in pleurisy it may be impossible to say where the dulness of the pleuritic effusion ends and the hepatic dulness begins, and here, as in some forms of true hepatic enlargement, there may be bulging of the ribs and obliteration of the intercostal spaces. Under such circumstances, there are several characters of considerable importance in diagnosis—viz. :

a. The bulging of the side of the chest is more uniform in pleurisy, and not abruptly limited to the lower part, as in diseases of the liver.

b. In pleuritic effusion, the upper margin of the dull space is horizontal (fig. 7), instead of arched as in enlargements of the liver.

c. In pleuritic effusion, the upper level of the dull space will often be found to vary with the position of the patient. In enlargement of the liver, it is the same in all positions.

d. In pleuritic effusion, the lower margin of the liver is not depressed upon deep inspiration, which is the case in enlargements of the liver, unless there be firm adhesions to the diaphragm.

e. Eversion of the lower right costal cartilages would indicate hepatic enlargement, and not pleuritic effusion.

f. When there is sufficient effusion into the pleura to cause downward bulging of the diaphragm, a depression may be sometimes observed between the lower margin of the ribs and the upper surface of the liver, which is not met with in hypertrophy of the liver.

Effusion into the pericardium will be recognised by the outline of the area of dulness on percussion. It is the left lobe of the liver that is mainly displaced by it.

In arriving at a diagnosis, it must not be forgotten that inflammation of the pleura or of the base of the right lung may coexist with enlargement of

the liver. This is a not uncommon occurrence in hydatid tumours or abscesses of the liver, and often precedes their bursting upwards through the diaphragm. So also after an hydatid tumour of the liver has burst into the pleura, extensive empyema may coexist with great enlargement of the liver. I shall hereafter have an opportunity of bringing under your notice the particulars of cases in which this occurred.

VI. *A tumour or collection of fluid between the upper surface of the liver and the diaphragm* may also cause great depression of the liver, and apparent enlargement of the organ. The upper margin of hepatic dulness will be arched, and it will probably be impossible during life to distinguish the case from one of real enlargement of the liver. You will find a case recorded by the late Dr. Bright, where a large abscess situated between the diaphragm and the liver produced apparent enlargement of the liver;* and more than once I have known enlargement of the liver simulated by an encysted collection of peritoneal fluid between the liver and the diaphragm, when the organ was in reality atrophied. Such cases, however, are rare.

VII. *Various abnormal conditions of the abdominal viscera* may displace the liver upwards, so that it encroaches upon the cavity of the chest, and appears enlarged. This happens not unfrequently in cases of ascites, and in ovarian and uterine tumours, in

* Clinical Memoirs on Abdominal Tumours. Syd. Soc. ed. p. 257.

aneurism of the abdominal aorta,* &c.; and hence elevation of the liver above its usual height must not, under such circumstances, be regarded as a sign of enlargement. Greater difficulty, however, in diagnosis may result from tumours in the omentum or in the right kidney, being in the immediate proximity of the liver, and appearing to be tumours of the liver itself. The difficulty will be increased if such tumours compress the common bile-duct, so as to occasion jaundice. The diagnosis of an omental tumour under such circumstances must mainly depend on the want of all uniformity in the apparent hepatic enlargement, the dimensions of the liver in every other direction being normal; while in tumours of the kidney the urine usually presents important changes, and at the same time, when the patient lies on his back, the finger can usually be inserted between the ribs and the upper part of the tumour.

Accumulations of fæces in the transverse colon also constitute a condition which it is often most difficult to distinguish from enlargement of the liver. Such cases are constantly occurring in practice, and it is important to bear in mind that, if you are to rely on the patient's statements, these accumulations are far from being necessarily associated with constipation. The resemblance to hepatic disease in these cases may be further increased by the hardened scybala imparting to the tumour a nodulated character like that of cancer, and by the development of such sym-

* Stokes. Op. cit. p. 617.

ptoms as jaundice, vomiting, and hiccup. The diagnosis of these cases from true enlargement of the liver must rest mainly on—

1. The occurrence of spasmodic pains like those resulting from obstructed bowels, &c.

2. The disappearance of the tumour, and the amelioration of the symptoms under such treatment as poultices and fomentations, purgatives, enemata, and belladonna.

Lastly.

VIII. *Abnormal conditions of the abdominal parietes* may simulate enlargements of the liver.

Firm contraction of the bellies of the recti muscles, owing either to inflammation of the subjacent peritoneum or stomach, or, in cases of increased muscular irritability, to the mere application of the hand, is frequently mistaken by inexperienced observers for hepatic enlargement. It is distinguished by:—

1. The situation, size, and form of the apparent tumour corresponding to those of the recti.

2. The sound on percussion being usually more clear and tympanitic than it would be over a solid tumour.

The diagnosis may also be considerably embarrassed by an inflammatory swelling in the abdominal parietes over the liver. This has often been mistaken for an abscess of the liver itself. Not long since a remarkable instance of this sort came under my notice in a patient recovering from typhus fever with parotid buboes. For some days the diagnosis was very

doubtful. The following characters usually suffice to distinguish this condition from hepatic disease :—

1. The margin of inflammation and of dulness on percussion is ill-defined, and does not correspond to the boundary of an enlarged liver.

2. There is a greater amount of hardness and tightness of the superimposed integuments.

3. The constitutional symptoms are comparatively slight, and there are no indications of hepatic derangement.

Keeping in view these sources of fallacy, we may now proceed to consider the various causes of true enlargement of the liver.

LECTURE II.

ENLARGEMENTS OF THE LIVER.

TRUE ENLARGEMENTS OF THE LIVER: SUBDIVISION INTO PAINLESS AND PAINFUL: 1. THE WAXY, LARDACEOUS, OR AMYLOID LIVER; 2. THE FATTY LIVER; 3. SIMPLE HYPERTROPHY.

BEARING in mind the various circumstances under which I have told you that hypertrophy of the liver may be simulated during life, we are now prepared for considering those cases in which an increased area of hepatic dulness is due to real enlargement of the organ. And first of all it may be observed that enlargement is a character common to many different diseases of the liver, so that some classification will be a material aid in diagnosis. The late Dr. Bright, whose researches on diseases of the abdomen are scarcely less valuable than those on diseases of the kidney, with which his name will for ever be associated, divided enlargements of the liver into two classes, according as their form was *smooth*, or *irregular*.* But this subdivision is, in my opinion, objectionable, inasmuch as in certain diseases (e.g. waxy liver) an enlargement which is usually regular and smooth, may assume a lobular or nodulated character, whereas in

* Abdominal Tumours. Syd. Soc. ed. p. 242.

others (e.g. cancer) an enlargement which is for the most part nodulated, may occasionally be perfectly smooth. A subdivision which appears to me to be, on the whole, preferable, is that into *painless* and *painful* enlargements. Painless enlargements are further characterised by an absence of jaundice, and a very chronic course; but in painful enlargements jaundice is a very common symptom, and the progress is more rapid.

Among painless enlargements we have the so-called amyloid liver, the fatty liver, hydatid tumour of the liver, and simple hypertrophy.

Among enlargements in which pain is a prominent symptom we have congestion, catarrh of the bile-ducts, obstruction of the common duct and retention of bile, pyæmic abscesses, tropical abscess, and cancer.

There are probably other enlargements of the liver besides those now mentioned, the anatomical and clinical characters of which are still unknown; but the distinguishing characters of the several forms of enlargement with which we are acquainted may now be considered in detail.

I. THE WAXY, LARDACEOUS, OR AMYLOID LIVER.

The liver undergoes greater enlargement from the so-called waxy, or amyloid deposit, than from any other disease, excepting, perhaps, cancer. I have known the liver of an adult affected with this disease weigh upwards of 180, instead of 50 or 60 ounces, and the liver, of which I show you here a portion,

weighed one-seventh, instead of a twenty-fifth, of the entire body of the child from whom it was taken. Enlargement of the liver due to waxy or amyloid deposit may be recognised during life by the following characters:—

1. The enlargement is often great, so that the liver fills up a large portion of the abdominal cavity.

2. It is uniform in every direction, so that the form of the organ is not essentially altered. The area of hepatic dulness on percussion is increased in the median, dorsal, and axillary lines, as well as in the right mammary. The increase is greater in front than behind, because in the former situation there is greater room for growth (figs. 9 and 10). It is increased in an upward as well as in a downward direction, although mainly in the latter, the lower margin often reaching the umbilicus, or even the right groin; but nowhere is there any outgrowth from the normal contour. The abdomen is enlarged, and often there is a visible tumour below the right costal arch and in the epigastrium, but rarely, if ever, is there any bulging of the ribs themselves. Waxy enlargement of the liver moulds itself over adjacent organs, and has little tendency to cause displacement of the ribs by excentric pressure.

3. On palpation, the portion of liver which extends below the margin of the ribs is very dense, firm, and resistant. There is no elasticity, and still less any feeling of fluctuation.

4. The outer surface is smooth, and the lower mar-

gin is more rounded than natural, regular, and free from all indentation. In this respect, however, rare exceptions occur, an ignorance of which may lead to

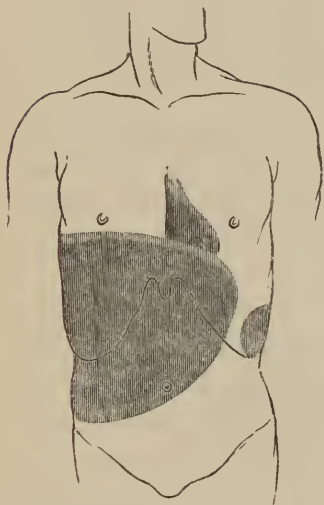


Fig. 9 shows the increased area of hepatic and of splenic dulness in the case of Henry D——: anterior view. Between the two is a space yielding the clear tympanic sound of the stomach; and above the liver is the normal area of cardiac dulness. Compare this with fig. 3, which shows the normal boundaries of the liver and spleen.



Fig. 10 shows the increased area of hepatic dulness in Henry D——: view on right side. The upper border is arched, and gradually falls towards the spine.

errors in diagnosis. In rare cases waxy deposit in the liver coexists with cirrhosis, or with what are

known as syphilitic cicatrices, and then the surface of the organ may be nodulated, or even broken up into irregular lobes, separated by deep fissures, the existence of which may lead to the suspicion that the enlargement is due to cancer. In cases also of extreme enlargement there may be an exaggeration, so to speak, of the lobes into which the liver is naturally divided, deep fissures corresponding to the attachment of the ligaments. Some of you had an opportunity of observing a case of this sort under the care of my colleague Dr. Greenhow, the particulars of which I shall relate to you presently. Cases have also been recorded by Professor Frerichs of Berlin, in which a waxy liver has presented a more or less lobulated form.

5. Waxy deposit in the liver has but little tendency to obstruct the portal circulation, and consequently ascites and enlargement of the subcutaneous veins of the abdominal wall are not common phenomena in its clinical history. When such indications of portal obstruction do occur, they are often due to pressure exerted on the trunk of the portal vein by lymphatic glands in the fissure of the liver enlarged from waxy deposit. Occasionally, also, fluid is effused into the peritoneum as the result of secondary peritonitis.

6. Jaundice is a rare symptom in waxy disease of the liver; and when it occurs, it is due, for the most part, to the pressure on the bile-ducts of enlarged lymphatic glands, or to the co-existence of catarrh of the bile-ducts.

7. Pain and tenderness are never prominent symptoms. The liver can be manipulated with impunity, and the patient complains only of a feeling of weight or tightness in the right hypochondrium, or of uneasiness from the pressure to which the stomach and intestines are subjected. But occasionally, and particularly where there is a syphilitic history, there is an attack of acute pain from intercurrent peri-hepatitis. In a case now under your notice, the enlargement commenced in India, with severe pain in the right side, for which numerous leeches were applied; but the enlarged liver now exhibits its usual painless character. Frerichs also records a case where the disease supervened on protracted ague, and where 'the first symptom was persistent cutting pains in the side.'*

8. The growth of the tumour is slow and imperceptible. It usually extends over several years.

9. Constitutionally, the symptoms are chiefly those of anæmia. There is no pyrexia; but the countenance is pale and sallow, the patient suffers from general debility, and the proportion of white corpuscles in the blood is slightly increased.

Other characters of no small moment in diagnosis, are derived from the spleen, the kidneys, the stomach, or the intestines being the seat of a similar morbid deposit to that producing the hepatic enlargement.

10. The spleen in most cases is enlarged, and often greatly, as well as the liver.

* Diseases of the Liver, Syd. Soc. Transl. vol. ii. p. 200.

11. Waxy disease of the kidneys has peculiar characters of its own, the presence of which in any case of hepatic enlargement would alone make it very probable that this enlargement was due to waxy deposit. These characters are:—

a. An increased quantity of urine. Not uncommonly the patient voids three or five pints of urine in the twenty-four hours. This is the rule throughout the greater part of the course of the disease. Towards the termination only is the quantity diminished.*

b. The urine is pale, of moderately low specific gravity (about 1012), free from any smokiness, and contains a considerable amount of albumen.

c. Casts of the renal tubes are often absent. When present, they may be of an epithelial or hyaline character, usually the latter, and most of them, from their size, appear to have come from tubes not denuded of their epithelium. These hyaline casts, so far as my observation goes, never yield the so-called amyloid reaction with iodine and sulphuric acid; but, in exceptional cases, this reaction may be observed in some of the cast-off renal cells.

d. During the greater part of the disease, when the urine is increased in quantity, there is rarely a material diminution in the excretion of urea, and consequently the tendency to uræmia is much less than in other forms of kidney disease.

* To Dr. Grainger Stewart, of Edinburgh, we are mainly indebted for pointing out the characters of the urine in waxy disease of the kidneys. My own observations coincide with his in every essential point.

The persistent secretion of a large quantity of urine containing much albumen by a person who has never had general anasarca, will of itself warrant the presumption that the individual is suffering from waxy disease of the kidneys. In the contracted or gouty kidney there may also be no dropsy, and the quantity of urine may be increased; but then the specific gravity is remarkably low (often not exceeding 1002 or 1005), and albumen is present as a mere trace, or may be entirely absent.

12. The implication of the stomach and intestines in the waxy disease induces a tendency to vomiting and to obstinate diarrhœa from slight causes. Occasionally this diarrhœa is accompanied by tenesmus, and the patient may be thought to labour under dysentery; but post-mortem examination reveals no evidence of inflammation of the bowel. The diarrhœa appears to result from deficient absorption rather than from increased exhalation.

13. Here, as in many other maladies, the circumstances under which the disease usually makes its appearance are of considerable importance in diagnosis. Now there are certain conditions of the system which pre-eminently favour the advent of waxy disease. Among them may be mentioned the following:—

a. Long-standing purulent discharge, such as is particularly apt to happen in connection with diseased bone or joints, and after surgical operations when the wound does not readily heal.

b. Constitutional syphilis. In a large number of cases of waxy disease the patients have been the subjects of constitutional syphilis, which appears to act as a predisposing cause quite independently of its inducing disease of the bones or protracted discharges.

c. Tubercle of the lungs and of other organs must be regarded as a predisposing cause of waxy degeneration, although the enlargement of the liver common under such circumstances is oftener fatty than waxy. Of 52 cases of persons dying from tubercle, and whose autopsies I have recorded, the liver was fatty in 20, and waxy in 6, and in 3 of the 6 there was likewise caries of the bones. Still, of the 52 cases, 14 had waxy disease of either the kidneys, the liver, or the spleen, or 1 in $3\frac{5}{7}$. The proportion of tubercular males in whom waxy disease was found was more than double that of females. Thus, of 33 tubercular males, there was waxy disease in 11, or 1 in 3; whereas of 19 tubercular females, only 3, or 1 in $6\frac{1}{3}$, had waxy disease.

d. Many chronic diseases that seriously impair the general nutrition probably predispose to waxy degeneration, which has thus been met with as an occasional sequel of protracted ague,* dysentery, cancer, &c.

Treatment.—The following rules comprise those measures which experience has shown to be most useful in the treatment of waxy disease of the liver.

* See Lecture IV.

In many cases, unfortunately, when the disease is already in an advanced stage, and when the kidneys and intestines are involved in the waxy degeneration, all treatment is of little avail, and the patient dies of exhaustion, which may be often ascribed to the copious drain of albumen in the urine or to the occurrence of profuse diarrhoea (as in Case III.). But, on the other hand, in not a few cases, the progress of the disease appears to be arrested by appropriate treatment, and in some, as in Case I., there is good reason for believing that the waxy deposit may be in great measure removed.

1. *Prevention.*—The prevention of diseases in general has not yet received from the practical physician the attention which it deserves. The more we study the causes of disease, the more apparent it is that we possess a power in this direction which has hitherto been too much neglected. Bearing in mind the causes which we have found to lead to waxy enlargement of the liver, the means for its prevention will at once suggest themselves. First and foremost, it is always advisable to arrest as early as possible copious suppuration from any part of the body, and in particular from diseased bone, and, if necessary, to have recourse to surgical interference for this purpose. It may indeed be a question whether some of those operations which what is called ‘conservative surgery’ has of late years substituted for amputation, from entailing protracted suppuration, have not sacrificed the life of the patient to the endeavour to save his

limb. The death of the patient is ascribed to a bad constitution, which may, however, possibly be the result of internal disease engendered by the operation. In cases where the disease of the liver comes on in the course of phthisis, our treatment must be directed to the primary disease, and every means should be employed to arrest the purulent discharge from the lungs, the diarrhoea, and the exhausting sweats. Again, the symptoms of constitutional syphilis must always be met by appropriate treatment; and measures must be taken to prevent the condition of general cachexia, which is apt to supervene on such enervating disease as ague and dysentery. Lastly, it may be mentioned that in cases where there is a copious suppurative drain from the system, alkalies have been proposed as a means of preventing the waxy deposit. Chemistry is said to have shown that the waxy material is dealkalised fibrine; and it is argued that as a large quantity of alkali passes off with the pus, the waxy deposit may be prevented by restoring this alkali to the system.* Experience has yet to decide on the practical utility of this recommendation, which certainly deserves a trial. I have had no opportunity of watching its effects, but most of you must have noticed in the wards a marked improvement in cases of waxy disease, under the use of the mineral acids.

When waxy disease is already present, we must combat it by such measures as the following:—

* Dr. Dickinson, *Med.-Chir. Trans.* vol. 1. p. 55.

2. The diet ought to be of as nutritious a character as is compatible with the digestive powers of the individual. A moderate allowance of alcoholic stimulants is generally useful. Considering the anæmic condition of the liver, alcohol is less likely to be injurious than in most other enlargements of the organ. When the disease is not too far advanced, and when the means of the patient permit, removal to a mild and equable climate is generally advisable.

3. *Tonics*.—Most patients suffering from waxy disease derive benefit from the use of tonics, and particularly of the various preparations of iron, such as the perchloride and the iodide. In more than one case I have met with marked improvement under the continued use of nitric acid, in combination with such vegetable bitters as gentian or quinine. The external use of nitro-muriatic acid in the way to be described to you in a future lecture (Lect. IV.) also deserves a trial. Cod-liver oil is of questionable utility; Frerichs states that he has known cases where waxy liver was developed under its continuous use.

4. Iodine and its preparations are of undoubted utility in the treatment of waxy disease. No preparation, I believe, is superior in this respect to the tincture of iodine of the British Pharmacopœia, which may be given in doses of 10 or 15 minims, diluted, three or four times a day. You will remember the marked improvement, not only in the general symptoms, but in the size of the liver, which took place under its use in the case of H. D. (Case II.). The

preparations of iodine will probably be found most useful in those cases where there is a syphilitic history.

5. Budd* has recorded cases where a marked improvement with diminution in the size of the liver has occurred under the use of the salts of ammonia, such as the muriate and the carbonate. In one case where the muriate of ammonia was given in doses of from 5 to 10 grains three times a day, a great enlargement of the liver, which had existed for nine months, and was accompanied by emaciation, pallor, and irritative fever, and where mercury, iodine, taraxacum, and nitro-muriatic acid had been tried in turn without success, was entirely reduced.

6. In all cases of waxy liver, you must be on the look-out for complications, and meet them when they arise. Those which you have chiefly to expect are diarrhœa, vomiting, albuminuria, dropsy, and uræmia. The diarrhœa must be met by mineral and vegetable astringents with opium, and counter-irritation to the abdomen. Even in cases where the kidneys are involved, opium is less to be dreaded than in other forms of kidney disease. But not unfrequently the diarrhœa resists all treatment and cuts off the patient. Persistent vomiting also is a serious complication, and is unaffected by treatment. Ice, bismuth, hydrocyanic acid, and counter-irritation to the epigastrium are the most useful remedies. The albuminuria requires no special treatment apart from that of the diseased liver. Dropsy must be met by diaphoretics and diuretics, the liquor ammoniæ acetatis with warm

* Dis. of Liver, 3rd ed. p. 335.

baths, and the bitartrate or acetate of potash with digitalis. With these remedies it will be well to combine the salts of iron, such as the perchloride with the liquor ammon. acetat., or the acetate of iron with the acetate of potash. Drastic purgatives must always be given with caution in this form of dropsy, for fear of inducing uncontrollable diarrhœa. Lastly, in those rare cases where uræmia occurs towards the close of the disease, the remedies indicated are diaphoretics, diuretics, the vapour-bath, and, if necessary, a brisk purgative.

In illustration of the remarks now made I show you in the first place a portion of a liver which I removed from the body of a patient who died in this hospital some years ago, and in whom the clinical history and post-mortem appearances were as follows:—

CASE I.—*Caries of the Hip-joint—Waxy Liver weighing nearly one-seventh of the entire body—Waxy Spleen—Fatty Kidneys.*

II. L——, aged 7, was admitted into the Middlesex Hospital under the care of Mr. Shaw, on November 30, 1858, having suffered from disease in the left hip-joint for about nine months. He was emaciated and of marked scrofulous habit, the head and joints being large in proportion to the rest of the body. There was considerable pain in the left hip, increased on movement, so that he walked with difficulty. Soon after admission, abscesses opened in the neighbourhood of the left hip, and sinuses continued to discharge until his death on January 27, 1861. During life there was great tumidity of the abdomen, obviously due to enlargement of the liver, the lower margin of which extended to below the umbilicus, and the surface of which was dense, smooth, and painless. The splenic dulness was also increased, and the boy passed urine containing much albumen, but he had no dropsy. He was also

liable to intercurrent attacks of diarrhoea, and the tongue was preternaturally clean, red, and glazed.

Post-mortem examination.—The body was extremely emaciated, the joints being large in proportion to the limbs. The total weight of the body was only 31 lb. 3 oz., or 499 oz. avoird.; the length of the body was $3\frac{1}{2}$ ft. The abdomen was remarkably tumid and hard, particularly in the right hypochondrium. There was much swelling about the left hip-joint, with numerous sinuses passing into the bone. The left thigh was flexed forwards and immovable. The entire head of the left femur was absent, and the end of the bone was carious. The acetabulum was likewise diseased, the bone being exposed and carious, and at one part deficient, so that there was an opening into the pelvic cavity.

The head was remarkably large, its circumference being $21\frac{1}{2}$ in. The brain weighed $55\frac{1}{2}$ oz.; its structure was normal. Each of the lateral ventricles contained three drachms of serum, and at the base were two fluid ounces. The membranes were normal.

The heart and lungs were normal.

The liver was enormously enlarged and very dense. Its weight was 69 oz. avoird., or nearly one-seventh of the weight of the whole body, the normal ratio for a child nine years of age being only about 1 to 25. It reached as far as the umbilicus, and moulded itself over the different organs in its vicinity. Its tissue was very firm, so that the organ retained its form when laid with its convex surface on the table. Its external surface was perfectly smooth and free from all adhesions, but exhibited impressions of the adjacent organs. Its cut surface was of a greyish-pink colour and translucent, and presented a network of opaque yellowish streaks composed of fibrous tissue, apparently corresponding to the outline of the enlarged lobules, and enclosing the firm translucent material in its meshes. Iodine and sulphuric acid developed the so-called amyloid reaction in a marked degree. On microscopic examination, the hepatic cells appeared to be coherent into flat scales, and could not be isolated. The nuclei were distinct, but the outlines of the cell-walls were scarcely appreciable at many places, the nuclei appearing interspersed through a translucent homogeneous mass: at some places even the nuclei could not be distinguished. Towards the circumference of the lobules the cells were more distinct, and at some places contained an unusual amount of oil.

The spleen weighed $11\frac{3}{4}$ oz., and presented a dense, glistening

surface on section, which became deeply tinged when treated with iodine and sulphuric acid.

The kidneys were large, the right weighing 5 oz., and the left $5\frac{1}{4}$ oz. They were not at all dense, but, on the contrary, very flabby. Their capsules were non-adherent, and their surfaces were perfectly smooth and pale yellow, with a beautiful network of injected veins. The cortical substance was hypertrophied, pale yellow, opaque, and soft. The renal epithelium throughout the kidneys was loaded with fine molecules and oil-globules, and at many places the uriniferous tubes appeared blocked up with oil. Iodine and sulphuric acid produced a decided tinging of the minute arteries and the Malpighian bodies in the cortex.

The mesenteric and Peyerian glands were slightly enlarged, and the application of iodine to the mucous membrane of the bowel produced numerous brownish-red puncta, corresponding to the villi.

The co-existence of fatty kidneys with waxy disease of the liver and spleen, in this case, is worthy of notice. It is to be observed, however, that even in the kidneys the minute vessels yielded the so-called amyloid reaction.

You have all had an opportunity of examining the patient whose case I am now about to relate, and who is still (April 1867) under my care in Cambridge Ward.

CASE II.—*Constitutional Syphilis, followed by Symptoms of Waxy Disease of the Liver, Spleen, and Kidneys.*

H. D——, aged 28, was admitted December 27, 1866. As a young man, he appears to have enjoyed good health, and to have been temperate. But six years ago he contracted syphilis, followed by buboes, which were opened, and the scars are still visible in the groins. The wound soon healed up, discharging only for about two weeks. He does not remember having had sore-throat or pains in the bones. In 1858 he joined a cavalry regiment in India. With the exception of one or two slight attacks of diarrhœa, his health still kept good until about November 1864, when he was seized with pain in the right hypochondrium, which confined him to bed for six weeks. The pain was increased on taking a long

inspiration; and he had leeches and blisters applied. At the end of six weeks he returned to his duty; but his liver enlarging and his strength failing, he was discharged from the service, and arrived in England again in June 1865. Since his return to England he has been able to earn his living as a labourer; but he has suffered each winter from a cough, and expectoration occasionally slightly streaked with blood. Eight weeks before admission he lost his appetite and strength and was sent as a case of 'fever' to the London Fever Hospital, where he took mercury and iodide of potassium, with the object of reducing the size of the liver. On leaving the Fever Hospital, he came here. He does not remember having had any form of fever in India, and at no time of his life has he had dropsy in any part of his body.

On admission, the patient was thin and anæmic, and had a decided sallowness of the countenance, without any jaundiced tint of the conjunctivæ. Over the back were numerous small scars and copper-coloured discolorations. But what was most remarkable was the enlargement of the liver, the upper margin of which rose as high as the fourth intercostal space, while the lower margin reached as low as the lower edge of the umbilicus (see fig. 9, p. 25). The organ appeared large in every direction, its dimensions being as follows: In median line, $8\frac{1}{4}$ in.; in right mammary line, $9\frac{3}{4}$ in.; in right axillary, $6\frac{1}{2}$ in.; in right dorsal, $5\frac{1}{2}$ in. The upper margin of the hepatic dulness was arched (fig. 10), that in the axillary line being an inch lower than that in the right mammary; in the right dorsal line it rose to the eighth intercostal space, and from this it gradually fell towards the spine. There was no bulging of the ribs, and the portion of liver below the margin of the costal arch was very firm and resistant, not at all tender, and perfectly smooth. The only appreciable inequality was a transverse furrow situated three inches and a-half above the umbilicus, and apparently due to the pressure of some article of clothing. The lower margin of the liver was considerably depressed when the patient took a long breath, so that the surface of the organ was probably not adherent, or only slightly so. The dimensions of the spleen were likewise increased (see fig. 9); it did not project beyond the margin of the costal arch, but the dimensions of the splenic dulness were—vertically, $5\frac{1}{2}$ in., and transversely, $6\frac{1}{2}$ in., instead of 2 in. vertically and 4 in. transversely, as in the normal state. There was no evidence of ascites or of anasarca. The patient's appetite was bad; his tongue was coated with a

white fur, and for some weeks after his admission there was a tendency to vomiting and diarrhoea, there being from three to four relaxed motions daily. The patient did not complain of pain in the abdomen, except of occasional transient attacks, which appeared to be due to flatulence. His chief complaint was of weakness in his limbs.

Since admission, the blood and urine have been carefully examined. The blood was found to contain a slight but decided increase in the proportion of white corpuscles, while many of the red corpuscles were of irregular outline and had a tendency to tail. The quantity of urine voided daily has been ascertained for several weeks, and has been always considerably above the healthy standard: the average quantity has been from three to four pints, and occasionally there has been more than four pints. Its specific gravity has varied from 1010 to 1015; it has always contained a considerable quantity of albumen, but has been perfectly clear, of an amber colour, and without any palpable deposit. Microscopic examination for casts of the uriniferous tubes has for the most part yielded negative results; on one occasion a few small hyaline casts were detected.

For the first five days after the patient's admission there was slight febrile disturbance. The pulse ranged from 110 to 120; the temperature rose to $102^{\circ}\cdot4$; and moist and dry bronchial râles could be heard over the back of both lungs. The patient had also sleepless nights, but without any rigors or perspirations. Since then the pulse and temperature have been normal, and the patient has slept well; but a little coarse crepitus can generally be heard at the bases of the lungs. There is no evidence of heart disease.

The treatment up to March 13, 1867, consisted in mineral acids, bitter tonics, and a generous diet. At first, sulphuric acid and small doses of laudanum were prescribed, with the object of checking the diarrhoea. On January 9, nitric acid was substituted for the sulphuric, and was given with small doses of laudanum in the compound infusion of gentian. On February 8 the opium was omitted, and a grain of quinine substituted for the compound infusion of gentian. The diarrhoea, which had quite ceased, at once returned, but was again held in check by the restoration of laudanum to the mixture on February 13. Under this treatment the patient steadily and greatly improved. He had a good appetite, and was much stronger. His weight on admission was only 7 st. $10\frac{1}{2}$ lb.; but on March 13 he had gained 16 lb.

April 3, 1867.—On March 13 the nitric acid was discontinued, and fifteen minims of compound tincture of iodine substituted. Since then the patient has continued to improve. He has now gained 20 lb. since admission. There has been no diarrhœa, and the quantity of urine has diminished almost to the natural standard. There is no material change, however, in the size of the liver.

April 29.—The patient was discharged from the hospital to-day, greatly improved in strength and appearance. There was no diarrhœa, and the urine was of the normal quantity, with only $\frac{1}{20}$ albumen. The size of the liver had also greatly diminished, as will be obvious from the following dimensions. In median line, 6 in.; in right mammary line, $7\frac{1}{4}$ in.; in right axillary, $6\frac{1}{4}$ in. The vertical splenic dulness was only $4\frac{1}{4}$ in.

The circumstance of the enlargement of the liver, in this case, commencing in the tropics with acute pain, might be thought to indicate abscess; but opposed to abscess are, the duration of the enlargement, its uniform character, its great density, the absence of fluctuation, and the fact of the patient having been able to work as a labourer for more than twelve months prior to his admission into the Fever Hospital. On the other hand, the physical characters of the hepatic swelling, the enlargement of the spleen, the excretion of a large quantity of very albuminous urine without any history of dropsy, the tendency to diarrhœa, the condition of the blood, and the syphilitic history, all point to waxy disease as the cause of the enlargement. As regards the pain, also, it may be stated that Frerichs records a case of waxy disease of the liver, in which ‘the first symptom was persistent cutting pains in the side, and soon his strength diminished to such an extent that he felt it necessary

to give up his work. Almost at the same time he observed a swelling in the right hypochondrium and epigastrium.'

CASE III.—*Syphilitic Necrosis of Lower Jaw—Albuminuria—Pleurisy and Pericarditis—Waxy Liver and Kidneys.*

John R—, aged 38, was admitted under 'my care on December 17, 1867. Six or seven years before he had contracted syphilis, and four years before he had been confined to bed for three months with a painful affection in the joints, which he believed to have been rheumatism, and ever since he had been liable to pains in the bones and joints. Twelve months before he had been a patient in this hospital with albuminuria and slight œdema of the legs, and at that time the alveolar process of the right side of the lower jaw exfoliated. Ten weeks before admission, he had been seized with cough, dyspnœa, and pain in the right side of the chest.

On admission, the patient had an anæmic, chlorotic countenance, with slight general anasarca. The urine contained a very large quantity of albumen—about one-half—but no tube-casts; it was passed in considerable quantity, and had a specific gravity of 1015. There was absolute dulness over the whole of the right lung, with all the signs of pleuritic effusion. The cardiac dulness was also increased, but could not be isolated from that of the right lung; the sounds of the heart were feeble, but no abnormal murmur could be detected. Pulse 96. The tongue was clean and red; the breath was extremely offensive; there was no appetite, and frequent vomiting. The hepatic dulness extended downwards uniformly, about two inches below the normal boundary; above it could not be well defined from the dulness over the right lung. The portion of liver projecting below the right ribs was smooth and free from tenderness. Splenic dulness not increased. Patient suffered much from want of sleep.

Treatment proved of no avail in relieving the patient's condition. On January 2, profuse diarrhœa, with watery, very offensive motions, came on. This continued until the patient's death occurred, on January 7, by exhaustion rather than by coma.

On examining the body, there was great thickening with firm

adhesions of the right pleura in front; posteriorly the right lung was separated from the chest-wall by about thirty ounces of turbid fluid. The right lung was extremely dense from fibroid change. The pericardium contained about twelve ounces of turbid serum, and the surface of the heart was coated with a thick rough layer of rather firmly adherent lymph. The liver, spleen, and base of right lung were all firmly adherent to the diaphragm. The liver weighed 66 oz. It was extremely dense, and presented to the naked eye the appearances and chemical reaction of waxy deposit. The spleen was of natural size and rather soft. The kidneys were of about the normal size, their surfaces were slightly granular, the cortices were extremely dense and pale, and the straight vessels and Malpighian bodies exhibited in a characteristic manner the so-called 'amyloid reaction.' The mucous membrane of the small intestine was intensely injected, but exhibited no 'amyloid reaction.'

In the following case, which occurred not long ago in this hospital, the diagnosis was rendered difficult by the irregular, nodulated form of the enlarged liver. The case was under the care of Dr. Greenhow, and is recorded in the 'Pathological Transactions,' vol. xvi. p. 147.

CASE IV.—*Waxy Liver enlarged and nodulated, simulating Cancer.*

The patient was a baker, aged 33, at the time of his death, on October 12, 1864. No cause could be assigned for the disease, but a scar of doubtful nature was noticed in the right groin. He first came under observation about four months before his death, and although the liver was then about as large as when he died, it had never been the seat of pain or discomfort, and indeed the patient was unaware of the existence of any tumour in the abdomen until it was discovered during his examination at the hospital. The tumour extended from the right to the left side, so as to occupy both hypochondria. There was absolute dulness on percussion from the fourth right rib to an inch above the level of the umbilicus. The tumour was not in the slightest degree tender, and its general surface was perfectly smooth. A smooth

globular prominence in the epigastrium, however, simulated somewhat a deeply-seated hydatid tumour, while a nodulated border and ascites subsequently gave rise to the suspicion of cancer. Still, the absence of pain or of the usual phenomena of the cancerous cachexia negatived the supposition of cancer; while the density of the epigastric tumour, the enlargement of the spleen, and the condition of the urine were in favour of waxy disease rather than of hydatid.

A fortnight before the patient was first seen, his feet began to swell, and the anasarca gradually extended up to the thighs and scrotum. About two months before death fluid began to collect in the peritoneum, but the dropsy never invaded the arms or upper part of the body. The urine was copious, about three pints, and contained much albumen, but rarely any casts. At no time was there jaundice. Towards the last the patient became greatly emaciated, and he finally died exhausted.

The liver weighed $184\frac{1}{2}$ oz., and was in an advanced stage of albuminous or waxy disease, yielding a most characteristic reaction with iodine. The spleen and kidneys, and the lymphatic glands in the portal fissure, were also greatly enlarged, and had undergone a similar change. Both lobes of the liver were equally enlarged, but they were prolonged upwards and backwards, so as to leave a fissure five inches in depth at the posterior margin, corresponding to the attachment of the suspensory ligament. The anterior border was much thickened, and was also indented by two deep fissures, corresponding to the notches for the suspensory ligament and the gall-bladder, which imparted to it a lobulated character. On the upper surface, also, corresponding to the epigastrium, there was a semi-globular elevation three inches in diameter. The under surface was marked by deep depressions, corresponding to the right kidney and the spleen. The surface of the liver generally was smooth, but the capsule was much thickened, and superiorly adherent to the diaphragm. The stomach, intestines, and heart were normal.

II. THE FATTY LIVER.

The second form of painless enlargement of the liver is that which is due to the accumulation of oil,

or 'the fatty liver.' This form of hepatic enlargement has the following clinical characters:—

1. The enlargement may be considerable, but is rarely so great as that often attained by the waxy liver. It is not often that the anterior or lower border reaches down beyond the umbilicus, or even so far. Occasionally, however, the vertical hepatic dulness is increased out of proportion to the actual amount of enlargement, in consequence of the organ being so soft and flabby that it folds upon itself, so that the anterior margin is depressed, and a larger portion of the organ is brought into apposition with the abdominal parietes.

2. As in waxy disease, the enlargement is tolerably uniform in every direction and there are no circumscribed bulgings, so that the natural form of the liver is but little altered. There is no expansion or bulging of the lower ribs.

3. The enlarged liver is less resistant to pressure and of softer consistence than in the waxy disease. Owing to its flabbiness, it is easily pushed aside by the finger, and when the abdominal parietes are thin, its soft, doughy consistence may be readily appreciated.

4. The outer surface is smooth, and the lower margin rounded. Although fatty degeneration may coexist with cirrhosis, the liver under such circumstances is reduced in size, and the irregular surface is rarely appreciable during life. A lobulated enlarged fatty liver is rarely, if ever, met with.

5. There is no ascites or enlargement of the superficial veins of the abdomen. A large accumulation of oil in the liver interferes with the circulation so far as to lead to an anæmic condition of the liver itself, but not to such an extent as to cause ascites.

6. Even in extreme cases bile continues to be secreted, and its secretion is not arrested or impeded. Jaundice, therefore, is not a symptom of the fatty liver.

7. The same remark applies to pain. Fatty enlargement of the liver is painless from first to last. The organ can be freely manipulated with impunity, although in extreme cases the patient may complain of a feeling of weight or distension in the abdomen, increased by turning on the left side.

8. From the absence of symptoms, few opportunities are afforded of watching the growth of fatty enlargement of the liver, but this is always slow and imperceptible.

9. The constitutional symptoms of fatty liver are few and not characteristic, and those which have been noted are often due for the most part to co-existing fatty degeneration of other organs, and more especially of the heart. General debility and want of tone in the nervous and vascular systems are amongst the most prominent symptoms. The patient is easily tired, and bears depletion or the inroads of acute disease badly. The late Dr. Addison described a condition of the integuments which he believed to be pathognomonic of fatty degeneration of the liver. 'To the eye,' he says, 'the skin presents a bloodless,

almost semi-transparent, and waxy appearance. When this is associated with mere pallor it is not very unlike fine polished ivory, but when combined with a more sallow tinge, as is now and then the case, it more resembles a common wax model. To the touch, the general integuments, for the most part, feel smooth, loose, and often flabby; whilst in some well-marked cases all its natural asperities would appear to be obliterated, and it becomes so exquisitely smooth and soft as to convey a sensation resembling that experienced on handling a piece of the softest satin.* These appearances are chiefly met with in females, and although they are far from being invariably present, yet in most cases the countenance and general integuments are more or less pasty and anæmic, and sometimes the skin appears greasy from increased action of the sebaceous follicles. Patients with fatty liver also suffer often from dyspeptic symptoms, such as flatulence, hypochondriasis, irregular action of the bowels—usually constipation, but not unfrequently profuse diarrhœa from slight causes.

10. Enlargement of the spleen is rarely present. The portal circulation is not obstructed to such an extent as to lead to enlargement of this organ from stasis of blood; and the spleen is not liable, as in waxy disease, to a deposit of the same material as that which causes the liver to enlarge.

There are, however, certain other organs which are apt to undergo fatty degeneration as well as the liver, and the disease in each of these organs has

* Guy's Hospital Reports, First Series, vol. i. 1836, p. 479.

symptoms of its own, which, when present, will throw light on the nature of the hepatic enlargement. Thus—

11. When there is fatty degeneration of the heart, in addition to the signs already enumerated, there are often—

a. A very feeble, or even inappreciable, cardiac impulse.

b. Very faint, or even inaudible, cardiac sounds.

c. A very slow, or a quick, feeble, and irregular radial pulse.

d. Attacks of vertigo, syncope, or pseudo-apoplexy.

e. Dyspnœa on slight exertion, and a feeling of sinking at the epigastrium.

12. When there is fatty degeneration of the kidneys, in addition to the signs already enumerated, there will usually be—

a. Urine below the normal standard in quantity, oftener turbid than clear, containing much albumen, and depositing numerous oil-casts.

b. A tendency to general anasarca.

c. Extreme pallor and pastiness of countenance.

13. As in waxy disease of the liver, the diagnosis will often be materially aided by attending to the circumstances under which the enlargement occurs. Many different conditions of the system may give rise to fatty enlargement of the liver, but most of them may be referred to one of the following heads:—

a. Large accumulations of fat beneath the skin throughout the body, in persons who for the most part are large feeders and lead indolent lives. It

is in this condition that the heart is most likely to participate in the fatty change, and that you will expect to discover the symptoms of fatty heart already referred to. It is patients in this state who are most prone to die of rupture of the heart. In the 'Pathological Transactions' you will find several cases recorded in which patients died of rupture of the heart, and where not only was the heart found in a state of fatty degeneration, but the liver was enormously enlarged from fatty deposit, and there was a large accumulation of fat throughout the body.*

b. Alcoholism.—Persons who drink immoderately of ardent spirits, particularly if they take little exercise, are very subject to fatty liver. Of thirteen persons who died of delirium tremens, Frerichs found the liver very fatty in six. Of two fatal cases of delirium tremens in which an autopsy was made by me in this hospital some years ago, there was considerable fatty enlargement of the liver in both: in one the organ weighed eighty-three ounces; in the other ninety-six ounces. It is under these circumstances that the kidneys often participate in the fatty degeneration.

c. Phthisis.—The great frequency of fatty enlargement of the liver in persons suffering from pulmonary consumption has been already referred to under the head of the waxy liver (p. 30). In consumptive females it is much more common than in males. In this

* See particularly case by Dr. Quain, vol. iii. p. 262; and case by Mr. Pollock, vol. xv. p. 84.

disease, it is not a little remarkable that, while fat disappears rapidly from almost every tissue in the body, it should accumulate in such large quantities in the liver.

d. Other wasting diseases besides phthisis—such, for instance, as cancer,* simple ulcer of the stomach,† and chronic dysentery‡—are likewise often attended by fatty enlargement of the liver.

It appears, then, that fatty liver is met with under two opposite conditions: one, in which there is an increased supply of material capable of being converted into oil, and where fat often accumulates in all the tissues of the body; the other, in which there is a rapid absorption of fat from all the tissues, with consequent emaciation. Its mode of production in the former case is sufficiently obvious; in the latter, the blood becomes loaded with oily matters derived from the patient's own tissues, and this oily matter is separated from the blood in its passage through the liver. The impaired absorption of oxygen in phthisis, interfering with the proper metamorphosis of the oil, accounts for fatty liver being more common in pulmonary than in other wasting diseases; and the greater frequency of fatty liver in women may be accounted

* See case of cancer of the larynx, by Mr. C. Heath, *Pathological Transactions*, vol. xiii. p. 28; and case of extensive cancerous ulceration of groin, by Dr. Budd, *Diseases of Liver*, p. 299.

† Case by Mr. R. Robinson, *Path. Trans.* vol. iv. p. 133; and by Mr. H. Thompson, *id.* vol. vi. p. 186.

‡ Case by Dr. Bright, in *Hospital Reports*, vol. i. p. 117.

for by women having in general a larger quantity of fat to be absorbed.

Treatment.—It is not often that fatty enlargement of the liver causes such a derangement of functions as in itself to call for treatment. As a rule, treatment must be directed against the conditions in which the enlargement in question is known to occur.

1. When the disease is developed in persons who are large feeders and of indolent habits, the fat will usually disappear from the liver, as well as from the rest of the body, on the individual adopting an opposite mode of life. He must rise early and take active exercise in the open air, and live principally on lean meat, fish, and green vegetables, with light claret, hock, or plain water to drink, and avoid butter, fat, oil, fermented drinks, strong wines, and all substances rich in starch or sugar. Under such a regimen, the fat will not only disappear, but the nutrition of the muscles will be improved, and the patient's strength increased. In cases, however, where there is reason to suspect the existence of fatty degeneration of the muscular tissue of the heart, the change of regimen here recommended must not be too sudden, and its effects must be carefully watched, while caution must be exercised in withdrawing the accustomed allowance of alcoholic stimulant.

2. When fatty liver is the result of alcoholism, a simple withdrawal of the cause will usually be sufficient to effect a diminution in the size of the liver.

3. Alkalies, alkaline carbonates, or compounds of

the alkalies with the vegetable acids, in combination with some vegetable bitter, such as taraxacum or gentian, have generally been found useful for correcting the digestive derangements resulting from fatty liver; and if the bowels be constipated, recourse may also be had to occasional doses of the compound rhubarb, or colocynth pills of the Pharmacopœia, in combination with blue pill and extract of henbane. Eating large quantities of common salt with the food has sometimes appeared useful; and, when circumstances permit, it may be advisable to recommend a trial of the alkaline or saline mineral waters of Carlsbad, Marienbad, Kissingen, Ems, or Vichy.

4. The preparations of iron are often of great service in cases where there is marked anæmia, and those which are best suited are the *ferrum redactum*, the *ferri et quiniæ citras*, the *ferri et ammoniæ citras*, and the *mistura ferri aromatica*. The chalybeate mineral waters of Tonbridge or Moffat, or of Spa, Pyrmont, or Schwalbach, on the Continent, are also useful for the same object.

5. Lastly, when the disease appears in the course of phthisis, it rarely calls for any special treatment, but its presence is a contra-indication to the use of cod-liver oil, or other oleaginous remedies.

In the following case, I had several opportunities of demonstrating to you in the wards the clinical characters of the fatty liver. The absence of albuminuria or of enlargement of the spleen made it

improbable that the enlargement was due to waxy deposit.

CASE V.—*Acute Phthisis—Fatty Liver.*

Charles C——, aged 57, was admitted into the Middlesex Hospital, under my care, on June 11, 1867. He had enjoyed good health until about two months before, when he began to suffer from frequent cough, emaciation, and night sweats, and subsequently from diarrhoea. On admission he was very thin and prostrate, he had a frequent cough, with purulent expectoration; there was marked dulness for several inches below the right clavicle, and coarse moist râles audible over the whole of both lungs. The bowels were very relaxed. The liver was much enlarged; the hepatic dulness in the right mammary line measuring 7 in., and reaching fully 3 in. below the margin of the ribs. The enlargement was uniform; its outer surface was smooth, but much softer and less resistant than that of the waxy liver, and it was devoid of all pain or tenderness. There was no jaundice, albuminuria, or enlargement of the spleen. The patient rapidly sank, and died on June 16.

On examination of the body, both lungs were found infiltrated throughout with yellow tubercle, breaking down at the apices into small cavities. At the right apex the pulmonary tissue had entirely disappeared. Numerous small ulcers, without tubercular deposit in large intestine. Kidneys and spleen healthy. Liver much enlarged, weighed seventy-eight ounces, was smooth, pale yellow, opaque, and extremely friable; the secreting cells throughout were loaded with oil.

III. SIMPLE HYPERTROPHY.

By 'simple hypertrophy' is understood an enlargement of the liver, due to an increased size of the lobules and an increased size or number of secreting cells, without any alteration of structure. The enlargement of the liver is uniform and rarely great; and, as might be expected, it is not attended with

any prominent symptom. The condition is comparatively rare, and has still to be studied. It has chiefly been observed in

a. Leukæmia; and in

b. Exceptional cases of saccharine diabetes.*

Hence, when the liver is found enlarged in either of these maladies without any obvious derangement of its functions, simple hypertrophy may be suspected. It has been suggested that the enlargement of the liver arising from protracted residence in hot climates may be of this nature; but in most cases this is probably due to chronic hyperæmia or to waxy disease (see Lecture IV.).

* See Frerichs' Diseases of Liver, Syd. Soc. Transl. vol. ii. p. 210. According to Budd, the liver in diabetes is often unusually small, and the lobules shrunken, from the quantity of oil being below the normal standard (Diseases of Liver, 3rd ed. p. 310).

LECTURE III.

ENLARGEMENTS OF THE LIVER.

IV. HYDATID TUMOUR.

THE fourth and last form of painless enlargement of the liver is that which is due to the presence of hydatid tumour. This is known during life by the following characters:—

1. The enlargement may be very great, so as to fill the greater part of the abdominal cavity, or reach upwards to near the clavicle, but in its earlier stages the hydatid may form a perceptible tumour at one part of the liver, not larger than an orange.

2. Unlike any of the enlargements already considered, it is not uniform in every direction, but usually it follows one direction in particular; so that the natural form of the liver is greatly altered (figs. 11 and 12, pp. 84, 86). If it grow upwards, the natural arched outline of the upper boundary of hepatic dullness will be exaggerated; if it grow downwards, the lower boundary of hepatic dullness will be found to be natural at some places, while at others there is an abrupt protuberance or tumour (see fig. 11). Not unfrequently, it takes a lateral direction, and causes more or less bulging of the ribs; and then the disease

is apt to be mistaken for empyema, which is readily distinguished by the characters already enumerated (see page 17).

3. It is neither dense nor doughy, but elastic, or even fluctuating. If the hydatid be deeply seated, with much hepatic tissue separating it from the outer surface, the tumour will be only elastic; but if it approach near to the surface there will be distinct fluctuation, with a thrill as from fluid, on palpation. Occasionally there is the sign known as '*hydatid vibration*.' This is a peculiar trembling sensation, experienced when three fingers of the left hand are laid flat on the tumour, and the back of the left middle finger is struck abruptly with the point of the middle finger of the right hand. This sign is not due to the presence of secondary cysts in the interior of the parent, nor is it peculiar to hydatid tumours. It is elicited when any large cyst, with thin tense walls and watery contents, is treated in the manner above described. But, inasmuch as the only tumours in the liver answering to these characters are hydatids, the sign referred to, when present, is of considerable value in the diagnosis of hydatids in the liver. Unfortunately, in a large proportion—probably the majority—of cases of hydatid tumours of the liver, it is altogether wanting.

4. The surface of the tumour is smooth, and free from irregularities of every sort. In rare cases, when there are several distinct cysts projecting from the surface of the liver, this organ may appear through

the abdominal parietes to have somewhat of a lobulated character, which may occasion considerable embarrassment in diagnosis. The possibility of this source of fallacy arising must be kept in view.

5. Ascites, œdema of the lower extremities, enlargement of the superficial veins of the abdomen, and hæmorrhoids are not distinguishing characters of hydatid enlargement of the liver. Their occurrence in rare cases must be regarded as in some measure accidental, and due to compression by the tumour of the trunk of the portal vein, or of the inferior vena cava. Care must be taken not to mistake for ascites an enormous hydatid tumour projecting down from the liver and filling the fore-part of the abdominal cavity. This is distinguished by a history of growth from above downwards, and by the portions of the abdomen yielding tympanitic percussion not being the most elevated in any position of the patient. For instance, when the patient lies on his back, there may be dulness on percussion and unmistakable evidence of fluid in the most elevated part of the abdomen, while in both flanks the percussion is tympanitic (see Case XIV.). When hydatid tumour of the liver coexists with ascites, and no opportunity has been afforded of examining the patient prior to the ascites, the diagnosis will be extremely difficult, if not impossible.

6. Enlargement of the spleen is not a common consequence of hydatid enlargement of the liver, but may occur under conditions similar to those which occa-

sion ascites. In very rare cases, also, the spleen may be enlarged from the presence of secondary hydatid tumours.

7. Jaundice is also an exceptional, and, so to speak, accidental symptom of hydatid enlargement of the liver. When present, it is due to pressure by the tumour on the common bile-duct, to catarrh of the bile-ducts, or to the bursting of the tumour into the ducts, which become obstructed by its contents. I show you here a specimen taken from the body of a gentleman under my care, in whom jaundice was due to the last of these causes (Case X.), and you have recently had an opportunity of studying the symptoms in a similar case, which proved fatal in the hospital (Case IX.).

8. Enlargement of the liver from hydatid tumour rarely interferes with the functions of the kidneys, and hence we do not meet with those alterations in the urine so common in waxy and of frequent occurrence in fatty enlargements. In rare cases, however, the kidneys also may be the seat of hydatids, or pyelitis may be induced by the pressure of a large hydatid tumour of the liver on the ureter. Under these circumstances the urine may contain large quantities of pus, as happened in a patient who was under my care in this hospital a few years ago, and the particulars of whose case I shall narrate to you presently (Case XVIII.).

9. The growth of an hydatid tumour is slow and imperceptible, and, when the tumour is large, it has usually existed for years before the patient has recourse

to medical advice. Dr. Budd mentions the case of a lady who died at the age of seventy-three, and in whose body two hydatid tumours of the liver were found, which there was reason to believe had existed since she was eight years old.*

10. The latent character of hydatid enlargement of the liver is one of its chief characteristics. It often attains a great size without causing any pain or uneasiness, and often indeed without the patient being aware of its existence. The first local indications of its presence are those resulting from pressure on adjoining parts, a feeling of weight or distension, or embarrassment of the breathing. Then, and not till then, it may become the seat of occasional attacks of acute pain and tenderness, in consequence of inflammation of the superimposed peritoneum; but, on the whole, even these attacks are rare.

11. There is, in like manner, an absence of all constitutional symptoms. Even when of large size, the tumour does not interfere with the functions of the liver. There is no pyrexia or impairment of the general health, and the chief symptoms are those due to pressure on adjoining organs, and interference with their functions. Some years ago a patient came to me complaining of cough and shortness of breath, and fearing that she was consumptive. On examining the chest, I found an enormous hydatid tumour of the liver compressing the right lung, and causing great bulging outwards of the ribs, as well as a prominent

* Diseases of Liver, 3rd ed. p. 433.

tumour in the abdomen. The patient had suffered nothing except the cough and dyspnœa, and was not aware of the existence of any tumour (Case XVIII.). Instances also are not uncommon of patients who have died from acute inflammation excited by the bursting of a large hydatid tumour of the liver, who, previous to the attack of fatal inflammation, have been thought to be in perfect health (Case XV.).

12. The diseases which are most readily confounded with hydatid of the liver are abscess, distended gall-bladder, effusion into the right pleura, aneurism, cancer, and cystic tumour of the kidney (see page 19 and also Case XXIII.).

a. The absence of symptoms, both constitutional and local, and the slow growth, of hydatid tumour, form a marked distinction between it and abscess, which, so far as its physical characters are concerned, is the form of hepatic enlargement most closely resembling hydatid. There is one source of fallacy, however, which must be kept in view, although an accurate diagnosis under the circumstances would not materially modify the prognosis or the treatment. An hydatid tumour of the liver occasionally inflames and suppurates, and then it presents all the constitutional and local phenomena of abscess. The diagnosis of this condition must depend entirely on the patient's previous history—the fact of a painless tumour having long preceded the symptoms of abscess, and the absence of exposure to the ordinary causes of tropical abscess.

b. A distended gall-bladder may closely resemble a pendulous hydatid of the liver, and may also be free from pain. It is recognized by its shape and position, by its development being usually accompanied by attacks of biliary colic, and by the fact that in most cases there is jaundice, owing to there being obstruction of the common duct.

c. Extensive effusion into the right pleura, with bulging of the ribs, and obliteration of the intercostal spaces, may closely simulate a large hydatid tumour; but, on the whole, an hydatid tumour of the liver is more likely to be regarded as an example of pleuritic effusion, than pleuritic effusion mistaken for hydatid. The hydatid is mainly distinguished by its insidious growth, and by the absence of constitutional symptoms. The chief physical distinction is derived from the upper boundary of the dull space. In pleuritic effusion this is horizontal (page 14); in hydatid tumour it is arched, being at a higher level in the infra-axillary space than it is close to the spine or near the sternum. The possibility of an hydatid tumour of the liver co-existing with pleuritic effusion must not be lost sight of (see Cases XV. and XVI.).

d. An aneurism of the abdominal aorta, or of the hepatic artery, may present a smooth, globular tumour, very like that of an hydatid. Its main distinctive characters are pulsation, bellows-murmur, and the fact that it is usually the seat of acute neuralgic pains, owing to pressure on the branches of the solar, or of the hepatic, plexus. An aneurism of

the hepatic artery is further distinguished by its being invariably accompanied by jaundice from compression of the bile-ducts.

e. Cancer of the liver is mainly distinguished by its irregular surface, tenderness and hardness, and by the absence of elasticity or feeling of fluctuation. The diagnosis may be embarrassed by the circumstance that several hydatid tumours projecting from the surface of the liver may impart to it an uneven surface, or that the nodules of medullary cancer may exhibit a degree of elasticity approaching to fluctuation. Under such circumstances, the diagnosis of hydatid must mainly depend on its slower growth and on the absence of constitutional cachexia.

If there be any doubts as to the nature of the case, they may at once be removed by an exploratory puncture. The fluid which escapes from an hydatid, even if it contain no echinococci or shreds of striated hydatid membrane, will reveal its nature with absolute certainty. If the sac be not inflamed, it is limpid, when running in a stream, with a slight opalescence when viewed in bulk; it is alkaline, and has a specific gravity of 1007 or 1009; it contains neither albumen nor urea, but throws down a copious white precipitate with nitrate of silver, owing to its strong impregnation with common salt. These characters apply to no other fluid in the body, whether healthy or morbid.* Even if the case should turn out to be

* The contrast between the fluid in the hydatid-cysts described in Case XX., and the surrounding peritoneal fluid, in which they were floating, is worthy of notice.

an aneurism or a cancer, no harm is likely to result from an exploratory puncture.

Modes of Termination of Hydatid Tumours of the Liver.—It may be thought that a tumour which causes so little inconvenience that even when of large size the patient himself may be ignorant of its existence, requires little interference in the way of medical treatment. In reference to practice it is therefore important to have a correct knowledge of the natural modes of termination of hydatid tumours of the liver. The chief of these are as follows :—

Spontaneous Cure.—In the first place, there can be no doubt that some of these tumours undergo a spontaneous cure. The parasite may die from calcification of the parent cyst preventing further growth, from inflammatory action lighted up by the entrance of bile or by some other cause, or from the secondary vesicles increasing out of all proportion to the fluid in which they float (Case XXII.); the parent cyst slowly shrivels up, and in place of the hydatid we find a putty-like material, the real nature of which is disclosed by its containing shreds of the striated hydatid membranes or hooklets of echinococci. But, unfortunately, this favourable result is confined for the most part to tumours of so small a size that they are not recognized during life. When the tumour is sufficiently large to give rise to symptoms and be diagnosed, such an event is so rare that it cannot be calculated on. The tumour then continues to increase in size. Its growth may be slow; it may extend over years; but almost as surely as the

tumour grows will it one day burst or lead to an equally certain, though less sudden, death. The directions in which an hydatid tumour of the liver may burst are very various, and the danger will vary accordingly.

1. *Into the Pleural Cavity or Pulmonary Tissue.*—This direction is more common than any other. It is almost always the right lung and pleura that are invaded. When the contents of the hydatid are discharged through an opening in the diaphragm into the pleura, acute and almost invariably fatal pleurisy is the result.*

If adhesions form between the diaphragm and the base of the right lung prior to the bursting of the hydatid, the contents of the latter may escape by the bronchial tubes, and the patient may recover; † but even here, in most cases, fatal inflammation or gangrene is set up in the lung, ‡ or the patient dies from exhaustion, owing to the profuse discharge from a large cavity, or from many small cavities, excavated in the lung. § Fatal pleurisy may result from an hydatid tumour of the liver, without any perforation of the diaphragm. || From Case XVII., also, it will

* See Cases XV. and XVI.; also Frerichs, *Dis. of Liver* (Syd. Soc. Ed.), ii. 235; Ogle, *Path. Trans.* xi. 299; Bristowe, *Path. Trans.* iii. 341; H. Davies, *Path. Trans.* i. 278; Davaine, *Traité des Entozooaires*, p. 437.

† For examples, see Bright, *Abdom. Tum.* (Syd. Soc. Ed.), p. 49; Todd, *Med. Times and Gazette*, Jan. 5, 1854; *Path. Trans.* iv. 44; v. 303; viii. 92; ix. 28; Davaine, *op. cit.* p. 449.

‡ See cases by Peacock, *Path. Trans.* ii. 72; Pollock, *id.* xvi. 155.

§ Frerichs, *op. cit.* ii. 264; Peacock, *Path. Trans.* vol. xv. p. 247; Davaine, *op. cit.* p. 443.

|| See Murchison, *Ed. Med. Journ.* Dec. 1865, Case XI., and case by Dr. Pollock, *Path. Trans.* v. 301.

be seen that an obsolete hydatid cyst of the liver may inflame, and, after establishing a communication with the bronchial tubes, may give rise to all the phenomena of gangrene of the lung.

2. *Into the Pericardium*.—This is, fortunately, a very rare direction, as the cases in which it has been noticed have been always fatal, either instantaneously by embarrassment of the heart's action, or within a few hours by acute pericarditis.*

3. *Into the Peritoneum*.—The tumour collapses, and violent and almost always fatal peritonitis is at once excited. This accident must not be confounded with the attacks of partial peritonitis, which are so common before the tumour bursts in any direction. The rupture of the sac is often caused by external violence, in the form of a blow, fall, or strain. In the museum of St. Mary's Hospital is the calcified cyst of an hydatid taken from the body of a man who dropped down dead after receiving a slight blow on the epigastrium from a comrade with whom he was sparring. The blow ruptured the cyst; the contents of the cyst escaped into the peritoneum, and the man died from shock. Three cases of fatal rupture in consequence of a fall are recorded by Mr. Cæsar Hawkins.† Three similar cases are mentioned by Frerichs; in two the rupture was caused by a fall, and in the third it was

* Two cases of rupture into the pericardium will be found in Davaine's work (p. 408); a third is recorded by Wunderlich (Med. Times and Gaz., Nov. 12, 1859, p. 488).

† Med.-Chir. Trans. vol. xviii. p. 124.

due to a strain; in one of the cases, death occurred within a quarter of an hour of the rupture. Eight additional cases have been collected by Davaine, in which death ensued within a few hours or days of the rupture of an hydatid tumour of the liver into the peritoneum. In several of the cases the rupture was caused by a fall or strain, and in one it occurred while the patient was wrestling with a comrade.* Rupture into the peritoneum was probably the cause of the fatal event in Case XX. On the other hand, Bright records a case where what appeared to be a large hydatid tumour of the liver burst into the abdomen, without being followed by a fatal result.† Ogle also mentions the case of a patient who recovered after the symptoms of peritonitis resulting from the rupture of an hydatid cyst in the omentum.‡

4. *Through the Abdominal Parietes or Lower Inter-costal Spaces.*—This is not a common mode of termination, although several cases are on record. The contents of the hydatid may be discharged by an opening at the umbilicus or in some other part of the abdominal parietes, or in one of the lower intercostal spaces, and the patient may get well. Even here, however, the cyst is apt to take on suppuration, and the patient may die from exhaustion or from peritonitis; or fatal hæmorrhage may occur from the interior of the sac, as in a case recorded by Dr. Bright. Of ten

* Davaine, op. cit. p. 493.

† Abdom. Tumours, Syd. Soc. Ed. p. 47.

‡ Path. Trans. xi. p. 295.

cases where a spontaneous opening occurred, and of which I have collected notes, five terminated fatally.*

5. *Into the Stomach or Intestine.*—This is the most favourable direction in which the tumour can burst, although death sometimes results from the peritonitis which is set up around the opening, or from secondary abscesses of the liver,† and unfortunately it is not a common mode of termination. According as the tumour opens into the stomach or the intestine, the hydatids are vomited or evacuated *per anum*;‡ sometimes they escape in both directions. The opening is usually small, so that the hydatids are discharged slowly.

Davaine has collected eleven cases where an hydatid tumour of the liver appeared to open into the stomach, of which six were fatal; and fifteen cases where there was reason to believe that it had opened into the intestine, of which only one was fatal. In one of Davaine's cases the tumour opened through the abdominal parietes, as well as into the stomach. In a case of large hydatid tumour of the liver which occurred in this hospital nine years ago, under the care of my friend Dr. A. P. Stewart, where the liquid contents were drawn off by a trocar, the tumour subsequently

* Budd, *Dis. of Liver*, 3rd ed. p. 437; Frerichs, *op. cit.* ii. p. 237; Hawkins, *Med.-Chir. Trans.* xviii. pp. 153, 158; Bright, *op. cit.* p. 50; Griffiths, *Lond. Med. Gaz.* 1844, vol. xxxiv. p. 585; Davaine, *op. cit.* p. 384, Obs. V.

† See a case under Dr. Owen Rees, *Med. Times and Gaz.* June 20, 1857.

‡ For examples, see Frerichs, *op. cit.* ii. p. 237; Budd, *op. cit.* p. 452; Bright, *op. cit.* p. 49; Davaine, *op. cit.* p. 496.

burst into the bowel, discharging numerous cysts *per anum*, and the patient made a good recovery. In the 'Gazette des Hôpitaux' for 1850, a remarkable case is recorded where three hydatid cysts of the liver opened spontaneously: the first, in 1833, into the bronchi; the second, in 1845, into the stomach; and the third, in 1848, into the intestine: the patient recovered.

Although hydatid tumours of the pelvis occasionally open into the urinary passages, I am not acquainted with any case where this has happened when the primary tumour has been in the liver.

6. *Into the Biliary Passages.*—It is not uncommon for a communication to be established between an hydatid tumour of the liver and one of the bile-ducts. In several cases where this has occurred, I have found the secondary cysts ruptured, empty, and more or less stained with bile. The entrance of bile, as was long ago stated by Cruveilhier, appears to be fatal to the life of the parasite, and in many cases probably constitutes the commencement of a spontaneous cure, while in other cases it lights up severe and even fatal inflammatory action in the cyst. (Cases IX. and XI.) Not only does bile enter the cyst, but occasionally the contents of the cyst pass into the bile-ducts and gall-bladder, causing obstruction of these passages, with persistent and often fatal jaundice. You have lately had an opportunity of watching a case of this sort

(Case IX.), and several others will be found in Davaine's work.* In the case which has been under your notice, the jaundice almost disappeared, although the stools remained colourless, in consequence of the bile draining away through the external opening. Mr. Hawkins has recorded a case where the common bile-duct was obstructed by hydatids, without jaundice, owing to the bile escaping by a fistulous opening into a bronchus.† But now and then the biliary passages become sufficiently dilated to permit the evacuation of the contents of the cyst through them into the bowel. This is an extremely rare occurrence, and most of the cases where it has been noticed have been fatal. A remarkable case is recorded by Dr. Hillier, where the contents of an hydatid tumour were discharged through the bile-duct into the bowel, but where the patient died in consequence of hæmorrhage from the wall of the cyst, the blood (derived apparently from branches of the hepatic artery) passing along the duct into the stomach and intestines.‡ Two cases are recorded by Dr. Wilks, where an hydatid cyst opened into a bile-duct, but where death was caused by peritonitis or by 'inflammation about the liver and ducts;' in one

* Op. cit. p. 462. In rare instances an hydatid tumour appears to be developed in the bile-duct, although the possibility of such an occurrence is denied by Davaine. Dr. Dickinson has recorded the case of an hydatid developed in the right hepatic duct, where obstruction of the common duct was caused by a portion of the cyst together with inspissated bile (Path. Trans. xiii. 104).

† Med.-Chir. Trans. xviii. p. 148.

‡ Path. Trans. vii. p. 222.

of the cases hydatid cysts had been vomited and passed from the bowel before the occurrence of inflammation.* Frerichs mentions a case where most of the contents of an hydatid had escaped by the bile-duct, but where the common duct ultimately became obstructed, and fatal rupture of the gall-bladder was the result.† Case X. is a very rare example of recovery after the discharge of the contents of a large hydatid cyst through the bile-duct into the bowel; but although the recovery appeared to be complete, several months afterwards the passage of some of the remaining contents of the tumour along the duct gave rise to severe pain and vomiting, and the muscular efforts in vomiting tore across some of the old adhesions: the result was fatal peritonitis.

The only other case of recovery, under like circumstances, which I have met with, is one referred to by Davaine, where there was reason to believe that an hydatid tumour of the liver had ruptured into the gall-bladder, and where the patient recovered after a severe attack of biliary colic and jaundice, accompanied by the passage *per anum* of both hydatid cysts and gall-stones.‡

7. *Into the Vena Cava Inferior.*—In exceptional cases, an hydatid tumour of the liver bursts into the inferior vena cava, and its contents, reaching the right side of the heart, become impacted in the

* Path. Trans. xi. p. 128.

† Op. cit. ii. p. 231.

‡ Op. cit. p. 477.

pulmonary artery and cause instant death. Three cases of this sort are mentioned by Frerichs.*

But, independently of rupture, there are various ways in which an hydatid tumour may destroy life.

1. *By Marasmus and Exhaustion.*—This was the mode of death in Case XIV., where an hydatid tumour of the liver became so large that the entire abdomen was enormously distended by it, and respiration was seriously embarrassed. This case was further remarkable from the circumstance that there were dulness and fluctuation over the greater part of the front of the distended abdomen, while the epigastrium and both flanks were tympanitic on percussion.

2. *By Pressure upon important Organs and Interference with their Functions.*—An hydatid tumour of the liver may compress the vena cava so as to cause anasarca and varices of the lower extremities,† or the portal vein, so as to induce ascites, and necessitate recourse to paracentesis.‡

By pressure upwards also it may embarrass the respiration and the action of the heart.

3. *By Suppuration or Gangrene of the Cyst, or Suppuration external to the Cyst, with or without Pyæmia, and Secondary Purulent Deposits.*—Cases IX. XI. XII.

* Op. cit. ii. p. 238. Two of these cases are related at greater length by Davaine (op. cit. p. 405).

† A case of this sort is recorded by Dr. Habershon, in Guy's Hospital Reports, 3rd ser. vol. vi. p. 182.

‡ See cases by Dr. Barker, Path. Trans. vol. vii. p. 225, and by Dr. Budd, Dis. of Liver, p. 451, and Hawkins in Med.-Chir. Trans. xviii. p. 149.

and XIII. afford illustrations of these modes of termination, and many similar cases are on record.* Bristowe has recorded a case where the secondary abscesses appeared due to obstruction of one of the ducts, † but in several of the cases pus has been found in the vein in the neighbourhood of the suppurating hydatid.

4. *By the Formation of Secondary Hydatid Tumours.*
—Secondary hydatid tumours may form in the liver or mesentery; ‡ and, if they be large or numerous, they may interfere with the patient's nutrition, and cause death by exhaustion, by peritonitis, or by uræmia from compression of the ureters, as in Case XIX. Not uncommonly they form in the lung, and destroy life by inducing pneumonia. Case XXI. is an instance of a secondary hydatid tumour compressing the spinal cord, and causing paraplegia.§ Dr. Barker relates the particulars of a case where death was due to the formation of a secondary hydatid tumour in the brain. || An interesting case is recorded by Dr. Wilks, of a girl, aged nineteen, who died suddenly, having previously been in good health. An hydatid tumour was found in the liver, and another at the apex of the left ventricle of the heart. The latter had burst,

* For examples, see Bright, op. cit. p. 37; Budd, op. cit. p. 444; and Frerichs, op. cit. ii. p. 245.

† Path. Trans. vol. ix. p. 290.

‡ See cases recorded by Bright, op. cit. pp. 13, 23, and 30; Jones, Path. Trans. v. 298; Peacock, id. xv. 247; Gibb, id. xvi. 157.

§ Another case of an hydatid tumour of the spinal column pressing on the cord is recorded by Dr. Ogle, Path. Trans. p. xi. 299.

|| Path. Trans. x. p. 6.

and discharged a loose hydatid into the cavity of the left ventricle. *

The treatment of hydatid tumours of the liver may be considered under the following heads.

1. Their *prophylaxis* is based on a knowledge of their cause. Hydatid tumours in man are developed from the eggs of a tape-worm which enter the body from without. This tape-worm, the *Tænia echinococcus*, the entire length of which does not exceed a quarter of an inch, inhabits the intestine of the dog and wolf, and is in no way connected with the pig, as is commonly believed to be the case. It has only four joints, and the ova are contained in the last, or proglottis, are voided with the fæces of the dog, and subsequently find their way into the human body with the food or drink. Arrived in the intestines, they are developed into embryos, which penetrate into the liver or other parts, in a way not yet satisfactorily explained, and are there developed into hydatid tumours.

But the ova of the *Tænia echinococcus* develop hydatids in other animals than man, and especially in the sheep. The hydatids of human beings, as Dr. Thudichum† observes, most frequently accompany them

* Path. Trans. xi. p. 71. See also Path. Trans. xv. p. 247. Cases of hydatid tumours of the heart, without any implication of the liver, are recorded by Habershon (Path. Trans. vi. p. 108), Budd (Path. Trans. x. p. 80), and Davaine, op. cit. p. 396. In Budd's case, an hydatid tumour at the apex of the heart had burst, and loose hydatids were found in the right ventricle and in the pulmonary artery.

† Report on Parasitic Diseases in Quadrupeds used as Food, in Seventh Report of Med. Off. of Privy Council, London, 1865.

to their graves, or, at all events, they are not permitted to continue their dangerous existence, but the echinococci of sheep are again set free in the process of slaughtering, and are devoured by dogs, to be again developed into tape-worms. While then, man does not contribute to the multiplication and propagation of echinococci, his constant liability to the disease is kept up by the cycle of infection which subsists between dogs and sheep.

It follows, therefore, that for the prophylaxis of hydatid tumours in man it is necessary:—

a. To prevent dogs feeding on the offal of sheep, and other animals infested with hydatids. Dogs ought to be rigidly excluded from all slaughter-houses or knackeries, and ‘dogs’ meat’ ought always to be thoroughly boiled.

b. To destroy, as far as possible, the tape-worms generated in the dog, for which purpose it would be well that all dogs were periodically physicked, and their excreta buried in the ground or burnt.

These are measures which are of national importance in such countries as Iceland, where the sheep-dog, during the long nights of winter, occupies the crowded dwelling of his master, and where hydatids are the cause of one-seventh of the human mortality, and which merit attention even in our own country.

2. *Medicines*.—It must be confessed that little or no dependence can be placed on any medicinal agent for effecting a change in the size or in the structure of an hydatid tumour. Among the many remedies

that have been proposed, common salt and iodide of potassium are the two which have been most relied on for destroying the life of the hydatid, but there is no evidence that either the one or the other is endowed with such a property. It is difficult to conceive how chloride of sodium can be unfavourable to the growth of an hydatid, when it is remembered how large a quantity of this salt is contained in the fluid contents of the cyst, and that, therefore, it must be compatible with, if not necessary to, the healthy existence of the parasite. And with regard to the preparations of iodine, there is not only no proof of their power to destroy the life of the parasite,* but there is positive evidence that the iodine does not reach it. Frerichs was unable to discover a trace of iodine in the fluid of an hydatid cyst, removed from a woman who had taken iodide of potassium for many weeks, and similar observations were made in Cases VI. VII. and VIII.

3. *Evacuation of the Fluid Contents of the Cyst by a*

* The following are references to instances in which iodide of potassium was thought to have effected the cure of an hydatid cyst:—Med. Times and Gaz. April 7, 1860, p. 344, and Feb. 17, 1855; Lancet, Oct. 16, 1858. In one, at least, of the cases, the disappearance of the tumour appeared to be due to its having burst. The others may be viewed in connection with a case related by Dr. P. McGillivray, where an hydatid tumour, which it was intended to tap, disappeared spontaneously, a few days after the patient's admission into hospital (Austral. Med. Journ. Aug. 1865). As Dr. M. remarks: 'If the patient had been getting iodide of potassium, common salt, or any other reputed specific, the medicine would, no doubt, have got the credit of the cure.' Certain it is that, in hundreds of cases, iodide of potassium has been taken in large quantities without producing the slightest change in the tumour.

fine Trocar and Canula, and Closure of the Opening.—

Although medicines be of little or no avail, there is, happily, one expedient which holds out a fair chance of effecting a permanent cure, and that is puncture of the cyst and removal of its contents. It is now many years since hydatid tumours of the liver were tapped by Sir Benjamin Brodie, and the patients made a good recovery.* Successful cases were afterwards published by Dr. Bright,† and by many other observers. It is only of late years, however, that the operation has been often resorted to, and even still it is very doubtful if most practitioners would not prefer leaving the patient to the very uncertain chances of a spontaneous cure, or would limit the operation to cases where the tumour is of a size rarely attained. The fears expressed are not unnatural, for in not a few cases the operation has been followed by dangerous symptoms or even death. The dangers of the operation are mainly two; viz. 1. Acute peritonitis, owing to the escape of a portion of the hydatid fluid into the peritoneal sac; and, 2. Suppuration of the cyst, owing, in some degree, to the admission of air.

These dangers have mainly arisen in cases where an opening has been made with a scalpel or a large trocar, on the mistaken supposition that it was necessary to remove the secondary cysts as well as the liquid, or because the tumour was thought to be an abscess.

But the dangers in question may be in a great

* Med.-Chir. Trans. vol. xviii. p. 119.

† Op. cit. p. 42.

measure avoided by employing a very fine trocar. Experience has shown that the removal of the liquid, which is as thin and limpid as water, suffices to destroy both the parent hydatid and its offspring, and accordingly this is all that is necessary to be done. The administration of chloroform before the operation is not advisable, as the pain is but momentary, and the vomiting sometimes induced by the chloroform interferes with that perfect rest of the parts which ought always to be insisted on for forty-eight hours after the puncture; but if the patient be young or nervous, it may be well to induce local anæsthesia by the ether-spray. The point selected for puncture ought to be that where the hydatid fluid appears to approach nearest to the surface. The injection, after removal of the fluid, of such substances as alcohol, iodine, oil of male fern, or bile, is unnecessary, and may be injurious, by exciting excessive inflammatory action. Care ought to be taken to prevent even the entrance of air, and for this purpose it is well to remove the canula before the whole of the fluid has been drawn off, or as soon as the fluid ceases to flow in a full stream, first passing a wire through the canula to ascertain whether the stoppage be due to the closure of its orifice by an hydatid vesicle. Dr. G. Budd * has further recommended, for the same object, that the fluid be drawn off by means of an exhausting syringe, adapted to the canula; but on one occasion (Case VI.) when I employed Dr. Budd's

* Med. Times and Gaz. May 19, 1860, p. 494.

apparatus, the patient experienced so much pain from the suction action of the syringe that I have preferred the simpler precaution above mentioned. After removal of the canula, the opening should be covered with a piece of lint steeped in collodion, over which a compress and bandage are applied, and for forty-eight hours the patient ought to be kept in the recumbent posture, and every movement of the body be strictly prohibited: it may be well also to give an opiate at once, and, if there be the slightest pain, this may be repeated after a few hours.

One advantage of using a fine instrument is that it is unnecessary to wait for the formation of adhesions between the tumour and the abdominal wall, or to endeavour to induce them, before puncturing. The walls of the cyst are so elastic that the small opening closes immediately that the instrument is withdrawn, and prevents all subsequent oozing from the interior. If there be no adhesions, however, one precaution ought never to be neglected, viz. during the removal of the canula to press the punctured portion of the abdominal wall against the cyst. By neglecting to do this the abdominal wall will be pulled away from the cyst in the extraction of the canula, and the fluid in the canula may drop into the peritoneum.

The patient often experiences immediate relief from the sensation of tension and other unpleasant symptoms, from which he may previously have suffered, and within three or four days he is up and walking about. Sometimes the operation is followed

by a feeling of uneasiness in the tumour, or by considerable pain and constitutional disturbance; but if the above rules be attended to, these symptoms soon pass off, and the patient makes a good recovery. It not unfrequently happens, however, that about a week or ten days after the operation the tumour appears again to enlarge. This enlargement is not due to a reaccumulation of the hydatid fluid, but to inflammatory products thrown out, probably between the collapsed parasite and the surrounding hepatic tissue, which are slowly reabsorbed. Under these circumstances it is well not to be hastily tempted to have recourse a second time to paracentesis. A certain degree of fulness may remain for many months, or even longer, in the site of the tumour, the existence of which has been cited as a proof that the operation has been unsuccessful. Yet inasmuch as the operation does not profess to remove the parent and secondary cysts, but only to kill the hydatid, and thereby avert those dangers which have been shown to result from its prolonged vitality, and induce that slow process of atrophy which sometimes occurs independently of an operation, the fulness referred to is only what might be expected. If by the operation we can prevent the dangers likely to arise from an hydatid tumour, nothing more is necessary.

The safety and efficiency of the operation now recommended may be regarded as established. You have had many opportunities of satisfying yourselves on this matter in the cases under my care and that

of my colleagues, in this hospital, during the last few years. In addition to the three cases which I have now brought under your notice (Cases VI. VII. and VIII.), I would particularly direct your attention to two others which were under the care of Dr. Greenhow, and which are reported in the eighteenth volume of the 'Pathological Transactions,' p. 127; in one of these the quantity of fluid drawn off amounted to 110, and in the other to 148 fluid ounces; fifteen months after the operation in the former case, the patient was free from all signs or symptoms of the tumour. These and other similar cases which might be quoted afford the best answer to the objection that the operation is only effectual where the tumour is of small capacity. It is true that the operation, in killing the parasite, occasionally excites a certain amount of inflammation between it and the cavity of the liver in which it is embedded which may favour the supposition that the sac is refilling, but in most cases this, after a short time, spontaneously subsides, and it is only in exceptional cases that a second operation for the evacuation of pus becomes necessary. I have collected the particulars of forty-six reported cases in which the operation was performed (see Table at page 118). In thirty-five of the cases the operation appears to have been perfectly successful: in ten cases it was followed by suppuration, necessitating a free opening; eight of these ten cases recovered, and two died, but in one of the fatal cases death is said to have resulted

‘not so much from the suppuration of the tapped cyst, as from the general prostration consequent upon the arrest of function of surrounding organs’ by the liver, which contained *three other* cysts, each holding a pint of fluid; and in the other it was occasioned by the bursting of the tumour into the lung *after* the establishment of a free external opening. In one case the patient died of peritonitis within twenty-four hours of the operation, but he was in a state of extreme prostration and emaciation before it was performed, and the propriety of having recourse to any operative procedure under the circumstances may be doubted. In estimating the results of the operation, those cases only of course ought to be taken into the calculation where it was resorted to as a curative measure, and those ought to be excluded where it was performed merely as a palliative, and where death was already inevitable. I have therefore excluded from the Table (appended to this lecture) several such cases, and others where the operation was performed with a large trocar, where caustic was employed to procure adhesions before puncturing, or where some irritating substance was injected after the withdrawal of the fluid, and also those where the hydatid had suppurated or been contaminated with bile before the operation. The operation here recommended is only adapted for those cases where the fluid retains its natural limpid character, and the results of other operative procedures ought certainly not to be confounded with it.

A careful consideration, then, of the whole matter—of the dangers of the disease when left alone, and of the inutility of medicines on the one hand, and of the success hitherto obtained from the operation on the other, leads to the practical conclusion that, in all cases where an hydatid tumour is large enough to be recognized during life and is increasing in size, it is advisable to puncture it at once. If the tumour appear to be diminishing in size, it may be well to wait, but it is unnecessary to wait for the formation of adhesions, or to endeavour to induce them. An hydatid tumour is not prone to form adhesions over its outer surface, like an abscess. By the time that adhesions form in the natural way, the tumour has attained a large size, and is probably eating its way into some of the adjoining cavities; the chances are increased of its becoming inflamed and converted into an abscess; its walls also are much less elastic than at an earlier stage, and a puncture through them will close up less readily, so that there is a greater risk of fluid escaping into the peritoneum after removal of the canula, if the adhesions be not sufficient to prevent it. While the walls are still elastic, the opening made by a fine trocar may be expected to close immediately that the instrument is withdrawn, and the existence of adhesions is therefore unnecessary.

4. *Evacuation of the Contents of the Cyst by a large Permanent Opening.*—In the case of Mrs. C. (Case IX.) you have had an opportunity of studying the dangers to which a person must be subjected who has a large

suppurating or perhaps gangrenous hydatid of the liver communicating by a free opening with the external atmosphere, and I have already pointed out to you that one-half of the cases where an external opening forms spontaneously are fatal. The dangers are mainly four: viz. *a.* Exhaustion from the protracted discharge; *b.* Pyæmia and secondary inflammations; *c.* Hæmorrhage from the cavity in the liver; *d.* Peritonitis. Of 62 cases of which I have collected the particulars, where an opening of this sort occurred spontaneously, or was made by caustic, by a large trocar, or by incision, 24 were fatal, and deducting those cases where the tumour had been previously punctured but had again closed, there remain 21 deaths to 22 recoveries. Many of those patients also, who ultimately recovered, endured a protracted and exhausting illness.

When, however, the symptoms, or an exploratory puncture, show that the sac has undergone suppuration, a large permanent opening is the only justifiable mode of operating. The opening should be made with a large trocar, and a silver canula or india-rubber tube secured in the wound until the whole of the hydatid contents have come away. The cavity ought also to be washed out two or three times a day with an aqueous solution of carbolic acid (2 per cent.). Before operating in this way, it will always be necessary to ascertain the existence of adhesions, and, if necessary, to produce them by an incision over the tumour, plugged with lint, or by the application of

caustic potash; or an opening may be made by successive applications of caustic potash, in the manner recommended by Récamier in cases of abscess.*

The records of the following cases may serve to impress upon you more forcibly the symptoms and the dangers of hydatid tumours of the liver, and their appropriate treatment.

CASE VI.—*Hydatid Tumour of the Liver—Paracentesis—Recovery.*

You have had an opportunity of studying the clinical characters of hydatid tumour of the liver, which have now been described, in the case of John N—, aged 28, who was admitted under my care on December 3, 1866. He was a clerk, and had been in the Crimea for fourteen months, in 1855 and 1856. His previous health had always been good. In September 1864 he had sore-throat and slight aching pain in his right side, and it was then discovered by Mr. Churton, of Erith, that he had a tumour in the epigastrium, which was almost as large then as when he came under your notice. After that he suffered no uneasiness in the tumour until February 1866, when it became the seat of occasional darting pains, and on this account he was a patient in this hospital, under my care, from March 31 to April 18, 1866. Excepting these pains, which were very transient, and unaccompanied by any tenderness, the patient's general health was good, and he had not the slightest fever. On April 7, an attempt was made to empty the cyst by means of a small trocar and canula and an exhausting syringe, the puncture being made to the *left* of the middle line, where the tumour was most prominent. The action of the syringe, however, caused much pain in the back and faintness, and the operation was abandoned after obtaining only four or five ounces of fluid, a quantity evidently much less than the tumour contained. Excepting an attack of urticaria, the operation was followed by no bad symptom.

The patient was readmitted on December 3, partly on account of

* Frerichs, Dis. of Liver, Syd. Soc. Ed. ii. p. 148.

a return of the slight pain from which he had previously suffered, but mainly with the object of having what was probably a second cyst emptied. At the time of his readmission, the following note was taken of his state:—‘Patient has a healthy appearance, and his only complaint is of a prominent tumour in the epigastrium, extending into both hypochondria, and evidently connected with the liver. It fills up the space between the sternum and the umbilicus, and causes a slight bulging of the ribs on both sides, particularly on the *right*. Its lower margin is about one inch above the umbilicus. It measures about 6 inches transversely, and 5 inches from above downwards. The hepatic dulness is

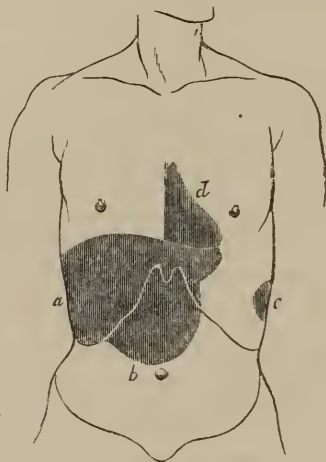


Fig. 11. Outline of Hepatic Dulness in the case of John N——, at the time of his admission into hospital, on December 3, 1866. *a*. hepatic dulness; *b*. tumour; *c*. spleen; *d*. heart.

6 inches in the mesial line, and 5 inches in the right mammary line; in the right axillary and dorsal lines it is normal. These dimensions exactly correspond with those noted when the patient left the hospital last April. The upper margin of hepatic dulness is not more arched than natural. The tumour is globular, perfectly smooth, and not at all tender. It is very elastic, distinctly fluctuates, and presents the character known as “hydatid vibration” in a marked degree. It does not appear to be adherent, as its

position varies with the respiratory movements. There is no jaundice, no ascites, no enlargement of the spleen, and no albumen in the urine. Tongue clean; bowels regular; no vomiting or pain after food; pulse 72.'

On December 7, Mr. Moore introduced a fine trocar into the most prominent part of the tumour, to the *right* of the middle line, and drew off by the canula, without any syringe, twenty fluid ounces of fluid. This fluid was opalescent, colourless, and alkaline, with a specific gravity of 1009; it contained no albumen, but yielded a copious white precipitate with nitrate of silver; numerous hooklets and several entire echinococci were discovered with the microscope. It is worthy of notice that, although the patient had been taking large doses of iodide of potassium for several days before both operations, on neither occasion did the fluid contain a trace of iodine.

The operation was not followed by the slightest febrile excitement or unfavourable symptom of any sort. On December 12, the patient got up, and on the 18th he left the hospital apparently well, the tumour showing no tendency to enlarge, and the hepatic dulness in the right mammary line being only $3\frac{3}{4}$ inches.

On March 18, 1867, I again saw John N——, who informed me that four days after leaving he was attacked with typhus fever, which he had probably contracted in the hospital, and with which he was dangerously ill. At the commencement of the fever the tumour appeared to enlarge, but by the time of his convalescence the swelling had quite subsided again, and now not the slightest trace of it can be discovered, the vertical hepatic dulness in the median line being only three inches.

March 9, 1868.—The patient presented himself at the hospital, and was examined by Dr. Thompson, Dr. Greenhow, Mr. Moore, and a large number of students, but no trace of a tumour could be discovered.

CASE VII.—*Hydatid Tumour of the Liver, threatening to burst—Paracentesis—Recovery.*

On August 3, 1864, Hannah S——, a very nervous woman, aged 31, consulted me about a tumour in the region of the liver. She was a cook in a medical man's family. In the summer of 1863 she had been laid up for three weeks with a pain across the stomach; but, with this exception, she had never suffered from any symptom of

abdominal disease until about nine weeks before she came to me. She was then seized suddenly with acute pain in the region of the liver, which lasted for about two hours. For several days she vomited after everything she ate, and she had great pain in her side when she attempted to cough or to turn in bed. She kept her bed for a week, and did not resume her work until after three weeks. The liver was then first observed to be enlarged and prominent, but the patient was unable to say whether this enlargement had existed before the attack of pain or not. On examination, there was found to be a slight bulging in the right hypochondrium below the ribs, this bulging being apparently continuous above with the liver, extending to half an inch below the umbilicus,

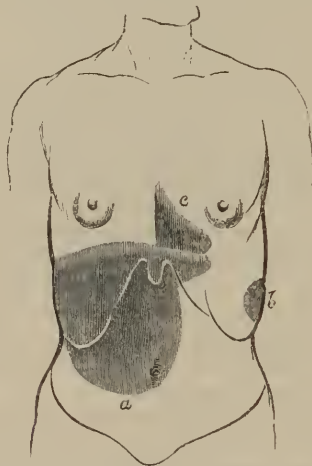


Fig. 12 represents the outline of Hepatic Dulness in the case of Hannah S—, in August 1864. *a.* tumour; *b.* spleen; *c.* heart.

and, transversely, from one inch to the left of the mesial line to about $3\frac{1}{2}$ inches to the right. The vertical hepatic dulness two inches within the right nipple was 7 inches, $4\frac{1}{4}$ inches of the dull space being below the edge of the ribs. The tumour was tense, but elastic and almost fluctuating. It was slightly tender on deep pressure. It did not appear to be adherent to the abdominal wall. Posteriorly, the hepatic dulness did not extend higher than usual,

and its upper margin was horizontal. The respiratory sounds at the right base were normal. The patient was slightly sallow, but had no decided jaundice. The tongue was clean, the appetite good, and the bowels regular. There was no ascites and no anasarca; the urine contained neither albumen nor bile-pigment. Pulse 84.

On August 7, the patient had a return of pain in the tumour, accompanied by vomiting and purging, lasting for two or three days. For several days after this attack the tumour was tender, and over its surface coarse friction could be both heard and felt during the respiratory movements.

On August 19, Hannah S—— was admitted, under my care, into Middlesex Hospital, and placed on iodide of potassium, five grains three times a day.

On August 24, the tumour was noted as more tense and tender. On the night of September 2, the patient had an attack of acute pain in right side, greatly increased by pressure, movement, or a long inspiration, and accompanied by much nausea, but by no vomiting or rigors. Pulse 96. Under the use of opium, poultices, and rest, these symptoms gradually subsided, but the tumour continued tender, the friction was again distinguishable for several days, and the pulse did not fall below 96. On September 9, the patient had another similar attack of pain, but more severe; the pulse rose to 116, and the friction returned. On September 14, the pain was less, but the tumour was observed to extend more to the right side, and was less rounded. On September 17, there was another severe attack of pain; and indeed, since August 24, the tumour had never been free from tenderness, while the patient felt herself gradually getting weaker, the pulse being rarely below 108.

Although there was no evidence of firm adhesions over the tumour, it was now determined to puncture it. From the first, the tumour had been diagnosed as an hydatid, and indeed the object of the patient's admission into hospital was to have it punctured. All who examined it were agreed that it contained fluid, and the only other affections at all likely to produce the appearances observed were a distended gall-bladder and an abscess of the liver. The tumour did not occupy quite the situation, and latterly did not present the shape of a distended gall-bladder, and there had never been jaundice. The persistent pain and tenderness noted for several weeks pointed rather to abscess, but there had been no rigors or perspirations, and, moreover, the tumour had not increased

much in size since it had been first observed. Supposing the tumour to be hydatid, there was reason to fear that it was about to burst.

On September 20, my colleague, Mr. Hulke, tapped the tumour with a fine trocar, the canula of which was scarcely so large as a No. 1 catheter, and drew off about twelve fluid ounces of clear limpid fluid, the specific gravity of which was 1009. No echinococci or hooklets could be discovered in it, but it was found to contain a large amount of chloride of sodium and no albumen. It did not contain a trace of any salt of iodine, although iodide of potassium had been taken almost continuously for several weeks.

In removing the canula, the abdominal parietes were pressed down against the tumour, and the puncture was afterwards covered with collodion and a pad. The patient was kept on her back for forty-eight hours, and not permitted to move. Twenty drops of laudanum were administered immediately after the operation, and for two days an opiate was given about once in four or six hours.

The night after the operation, the patient slept well. On the following day, the urine was retained, and was drawn off by catheter; and on September 22, the abdomen was distended and tympanitic, the skin hot and dry (temperature 101°), the pulse 120, and there was much thirst. Still there was much less pain and tenderness over the tumour than before the operation. The bowels had not been open for two days. An enema of turpentine and confection of rue brought away a large quantity of flatus, and the patient at once began to improve. On September 26, the pulse was 96, the tongue clean and moist, and the appetite was returning. For the first time for several weeks, the patient could tolerate free manipulation of the tumour, the dimensions of which were much reduced. On September 27, pulse 84; the collodion was removed from the wound, from which not a drop of discharge had escaped. On September 30, the patient was able to get up. Convalescence was retarded by an attack of facial rheumatism and other trifling ailments; but, on November 22, the patient was able to leave the hospital. The dimensions of the tumour were gradually diminishing, so that the dulness from the upper margin of the liver to the lower margin of the tumour did not exceed $5\frac{3}{4}$ inches. The tumour also was quite soft and free from tension, and could be manipulated without causing pain. The tongue was clean and moist; the appetite and digestion good. Pulse 100.

June 1867.—Nearly three years have now elapsed since the operation, and during the most of that time the patient has been able to follow her occupation as a cook, subject only to flatulence and other symptoms of dyspepsia and hysteria. Only a slight fulness is now perceptible in the epigastrium.

March 1868.—The patient writes that she is quite well, and is about to be married.

CASE VIII.—*Hydatid Tumour of Liver—Puncture with fine Trocar—Recovery.*

Elizabeth C—, aged 6, was admitted into the Middlesex Hospital under my care on December 3, 1867. With the exception of hooping-cough at the age of 3, she had always enjoyed excellent health; but her mother, almost since she was an infant, had noticed that she was larger about the waist than natural. Three months before, the girl had been seen by Miss Garrett, L.S.A., who diagnosed an hydatid tumour of the liver. Since then the mother thinks that the tumour has been increasing, but the only uneasiness the child has experienced has been an occasional feeling of sickness, a morning cough, and slight pain in the region of the liver. On admission, the patient was a robust, healthy-looking child, who seemed to have nothing amiss with her, with the exception of a swelling in the epigastrium, extending vertically from the lower end of the sternum to the umbilicus, and $2\frac{1}{2}$ inches laterally to either side of the median line. The tumour was globular, smooth, painless on manipulation, and with distinct fluctuation, and ‘hydatid vibration.’ It was quite movable over the subjacent parts, and did not appear to be adherent to the abdominal parietes, as it descended readily with inspiration. Although evidently connected with the liver, the area of hepatic dulness was not generally increased, its extent in the right mammary line measuring only $2\frac{1}{2}$ inches. The girth of the abdomen over the tumour was as follows:—

	Dec. 3	Dec. 20	Jan. 16	Jan. 24	March 9
At umbilicus	24·3	23·3	24·5	22·75	22·5
At ensiform cartilage	24·5	23·5	23·5	23·75	23·5
Half-way between um- bilicus and ensiform cartilage	25·75	24·66	25·25	24	22·5

The patient’s tongue was clean, her appetite good, and her bowels regular. There was neither ascites nor jaundice. Pulse 96.

She was ordered a draught containing two grains of iodide of potassium three times a day.

On December 10, Mr. Hulke punctured the tumour with a fine trocar, and drew off fourteen fluid ounces of fluid. This was colourless, slightly opalescent, with a specific gravity of 1010, and contained no albumen, but a large quantity of chlorides; neither echinococci, nor hooklets, nor any trace of iodine could be detected in it. Two hours after the operation the patient was sitting up in bed laughing and talking as if nothing had happened. During the following night, however, she had several attacks of vomiting (which was, perhaps, the effect of the chloroform that had been administered), and for two days the pulse rose to 140, and the temperature was as high as $100^{\circ}\cdot8$, but there was no tenderness of the abdomen, nor thoracic breathing.

On December 13, the temperature and pulse were again normal, and after this the patient had no bad symptom, except that from December 20 till January 14 the tumour appeared to increase again slowly in size, so that the question of performing paracentesis a second time was entertained. This, however, was abandoned, as the tumour began to diminish spontaneously, as will appear from the table of measurements. On March 9, there was no perceptible bulging, and scarcely any tumour to be felt.

In the following case, which you must all have watched with much interest, we were enabled to diagnose during the patient's life that a communication had been established between the tumour and the common bile-duct. The fact that the tumour had undergone suppuration contraindicated the ordinary operation, and compelled us to substitute a large permanent opening.

CASE IX.—*Hydatid Tumour of Liver, opening into the common Bile-duct.—Jaundice and Suppuration of the Cyst—Puncture with a large Trocar, and Permanent Opening—Pneumonia—Death.*

On February 4, 1868, I was requested by Mr. Ayling, of Great Portland Street, to see Mrs. C—, aged 30, who was suffering

from jaundice and enlargement of the liver. Her mother stated, that ever since she had been fourteen there had been a fulness in the epigastrium and left hypochondrium, but that, with the exception of occasional pain after food and other symptoms of indigestion, she had enjoyed good health until her present illness. She had been married for eleven years, and during that period the catamenia had been regular, and she had had no children or miscarriages. Eighteen days before I saw her, she had been suddenly seized with severe pain in the back and upper part of the abdomen, which almost bent her double. This was relieved by warm poultices, &c., but was soon followed by pyrexia, and four days later by jaundice, which soon became intense, with dark porter-coloured urine, and a complete absence of bile from the motions. The fever continued; the swelling in the epigastrium and left hypochondrium was observed to increase, and the patient was so prostrate, that some days before I saw her she was thought to be sinking; but she had no vomiting, rigors, or night-sweats.

I found the patient much emaciated, and with deep jaundice of the conjunctivæ and whole surface of the body. There was a distinct tumour in the epigastrium, extending apparently into both hypochondria. It projected forwards fully $1\frac{1}{2}$ inch beyond the natural level, and pushed forward the lower end of the sternum, and the lower ribs on both sides, but particularly on the left. When the patient lay on her back, the lower margin of the tumour was 1 inch above the umbilicus. The tumour was evidently connected with the liver, the dulness of which in the mesial line was 9 inches, in the right mammary line 5 inches, and in the left 6 inches. Posteriorly and laterally the hepatic dulness did not rise higher than natural on the right side, but on the left, posteriorly, it was fully 2 inches higher than on the right, and the dulness in the left axillary line was 9 inches. The tumour, where it presented itself at the epigastrium, was rounded, smooth, and slightly tender. Distinct fluctuation could be felt in it, and a thrill, as from fluid, could be made out in the epigastrium when percussion was made over the dull part at the back of the left side of the chest. The tongue was very red and clean, with enlarged papillæ at the tip, and the centre smooth and deeply fissured. The motions were clay-coloured, without a trace of bile-pigment. Pulse 108. Apex of heart elevated by tumour to between fourth and fifth ribs. Respirations 28, and slightly embarrassed, but pulmonary signs normal. Temperature $100^{\circ}6$. Urine 1027,

containing both bile-pigment and bile-acids (Harley's test), but no albumen.

The fact that the tumour contained fluid, and had probably existed for years without giving rise to symptoms, indicated hydatid; the acute pain, followed by jaundice, with disappearance of bile from the stools, made it probable that this hydatid had communicated with and obstructed the main bile-duct; while the enlargement of the tumour, with fever and great prostration, was accounted for by inflammation of the tumour, consequent on the entrance of bile. This was the diagnosis.

On the following day the patient was admitted, under my care, into the Middlesex Hospital, and as her condition became daily more critical, it was determined to have recourse to puncture of the tumour, as holding out the only chance of safety. Accordingly, on February 7, a fine trocar was introduced by Hulke in the left side of the epigastrium, and about six ounces of fluid drawn off. This was deeply tinged with bile and very fetid, and contained numerous pus corpuscles and scales of cholesterine, but no hooklets or echinococci. On ascertaining the nature of the fluid, the small canula was withdrawn, and a full-sized trocar substituted. Several hydatid vesicles escaped through the larger tube, but only about eight ounces more of fluid, although a probe could be passed in 6 or 8 inches. It appeared, therefore, that the contents of the cyst consisted mainly of hydatid vesicles. A solution of carbolic acid (2 per cent.) was injected into the cavity, and a large tube was tied into the wound.

During the ten days that followed the operation, several pints of the carbolic acid solution were injected three times a day through an elastic catheter passed into the cavity, and on each occasion large numbers of hydatid vesicles (with hooklets and echinococci in some) came away, with a fetid, purulent fluid, containing a large quantity of green bile. While this was going on, the abdomen returned to almost its normal dimensions, and the jaundice in a great measure disappeared from the integuments and urine, but the motions remained as light as before.

The patient had repeated doses of morphia after the operation, and for four days the pulse was about 108, the temperature was normal, and there was no very bad symptom, except the development on the tongue, and inside of the mouth of numerous aphthous ulcers on a raised base, which caused excruciating pain whenever she took food or drinks; but both the pain and the ulcers almost

entirely disappeared from repeatedly washing out the mouth with Condry's 'ozonized water.' During the night of February 11, the patient suffered from repeated rigors, and after this the pulse rose to 132 or 140, the respirations became quick, and the tongue dry; there was occasional vomiting, and the prostration rapidly increased. On the morning of the 18th delirium set in, and at 6 P.M. she died.

On opening the abdomen, the peritoneum contained no fluid, and there was no sign of recent peritonitis, but there were firm adhesions between the tumour and diaphragm and the abdominal parietes in front. The left lobe of the liver had disappeared, and its place was occupied by an enormous hydatid cyst. This cyst contained about two pints of very fetid thick green fluid, with large fragments of the parent hydatid cyst lying loose in the cavity. It opened externally through the wound in the abdominal wall, while internally it communicated with the common bile-duct by an opening large enough to admit a full-sized catheter. On slitting open the duodenum, the orifice of the duct was found sufficiently dilated to admit a goose quill, but obstructed by a large hydatid cyst, partially protruded into the duodenum. Between this and the opening into the cyst, the duct was distended with hydatid vesicles. The bile-ducts throughout the liver were greatly dilated and the liver itself was very fatty and intensely jaundiced, with a tight-lace prolongation downwards of the right lobe. There was no trace of bile-pigment in the intestinal contents. The spleen was adherent to the tumour, but otherwise normal; kidneys healthy. There was recent pneumonia, at some places passing into the condition of grey hepatization, of the back of the lower lobe of both lungs and of the upper lobe of the right.

The following case resembles in some respects that last recorded. It is remarkable no less for the fact that the patient recovered after discharging the contents of a large hydatid of the liver through the bile-duct into the bowel, than for the extraordinary manner in which death ultimately occurred (see page 69).

CASE X.—*Hydatid Tumour of the Liver, bursting into the Bile-duct—Jaundice—Discharge of innumerable Hydatid Membranes per Anum—Recovery—Attacks of Biliary Colic from passage of Cysts remaining in Liver through the Bile-duct—Rupture of old Adhesions of Liver during act of Vomiting—Peritonitis—Death.*

On October 29, 1861, I was consulted by Mr. G. W——, a solicitor, aged 53. For some weeks he had been suffering from flatulence and a feeling of tightness and oppression after meals, and three days before he had been attacked with severe pains in the abdomen, resembling colic. The countenance was somewhat sallow; the motions were pale, but contained bile; there was no bile in the urine, which was scanty and dark, having a specific gravity of 1027, and depositing much lithic acid. The vertical hepatic dulness in the right mammary line extended about an inch below the edge of the ribs, and all along the right hypochondrium there was slight tenderness on pressure. Pulse 64. His digestion had always been good, except once, about seven years before, when he had several attacks of colicky pain in the abdomen, similar to those from which he had recently suffered. The medicines prescribed by myself, and afterwards by Sir Thomas Watson, who met me in consultation, failed to give any relief.

On November 24, the patient had an attack of vomiting, followed by an aggravation of the dyspeptic symptoms, and by increased tenderness in the right hypochondrium.

On December 6, he was much worse. The tenderness in the right side had increased greatly, and there was also constant pain there, which became very acute when he took a long breath or coughed. The tongue was furred and moist. The bowels were very costive, and there was considerable tympanitic distension of the abdomen and increased sallowness, but no sickness. The pulse had risen to 88, and the respirations were 30, and thoracic. Fifteen leeches were applied to the seat of pain; twelve more on December 8, and eight more on December 10, with poultices in the intervals, and the bowels were kept open by castor-oil and turpentine enemata.

On December 12, the pain was much less, but there was still

considerable tenderness and a stitch in the right side on taking a breath or coughing. The countenance was very sallow, but there was no decided yellowness of the conjunctivæ, and the motions, though pale, contained bile. The vertical hepatic dulness in the right mammary line was 5 inches. Nothing like a defined tumour could be felt, and there was no bulging of the ribs. The breathing at the base of the right lung was normal. Pulse 88.*

On December 16 and 17, the patient passed, for the first time, several hydatid cysts in a bilious motion.

On December 18, he was much worse. There was decided jaundice of the integuments; the urine was loaded with bile-pigment, and there was not a trace of bile or of hydatid membranes in the motions. There was a constant pain in the right side, in addition to occasional paroxysms, like colic; lips parched; tongue furred; much perspiration in the night, and great prostration. Pulse 100. The treatment consisted in the constant application of poultices to the side, and in the administration of quinine, and of blue pill and opium.

December 19.—Is much easier. Has passed innumerable hydatid vesicles, from a pin's head to an orange in size, per anum. Skin and urine still jaundiced, and no bile in stools.

20.—Fæces to-day are tinged with bile, and still contain numerous hydatid cysts.

21.—Jaundice almost gone. Motions still contain hydatids and abundance of bile. Below and to the left of the right nipple, there is tympanitic percussion over a space the size of a crown-piece. Both above and below this there is hepatic dulness. Pulse 88; pain much less; tongue cleaning.

The patient continued to pass a few hydatid vesicles with each motion up to December 31, and the tympanitic percussion sound above noted remained a few days later than this. He had occasional sharp but temporary attacks of pain in the abdomen, resembling colic. On January 6, 1862, he was quite convalescent. Pulse 72. The tympanitic sound noted above could no longer be distinguished, and the upper border of hepatic dulness was an inch lower than before. At the end of January, Mr. W—— was able to drive out; and on February 19, he went to Ventnor for change of air, returning to London on March 11.

* From this date until January 25, I attended Mr. W—— in conjunction with Mr. F. Davies, of Upper Gower Street.

Once, while at Ventnor, he had a severe attack of colicky pain, lasting for an hour and a half, and 'bending him up double.' He had a similar attack, but less severe, a few days after his return to London. Both attacks were unaccompanied by vomiting. Every day he gained strength, and on his return to town he was able to resume his business. On April 2, he went down to Essex on business. He walked about the country several miles every day, feeling none the worse, and returned to town on April 6.

On April 8, he went to his business as usual, and walked several miles. Shortly after dinner, about 7 P.M., he was suddenly seized with severe pain in the abdomen, which returned in paroxysms, and this time was accompanied by vomiting. There was slight tenderness at the epigastrium, but no jaundice. The pulse was only 84. Repeated doses of opium and chloric ether were prescribed, and poultices were kept constantly applied over the abdomen.

On the following day, the paroxysms of pain had ceased, but there was more tenderness at the epigastrium and in the right hypochondrium, and considerable pain when he coughed or moved. The vomiting had not quite ceased. There was slight sallowness, but the stools contained bile. Pulse 86. Ten leeches were ordered to be applied to the side, and the poultices and opiates were to be continued.

The patient did not apply the leeches, as he felt better. In the afternoon, he had two severe attacks of rigors, after which he felt so much better and free from pain that he thought it unnecessary to send for me.

On the morning of April 10, he said that he felt so much better that he had eaten a good breakfast, and wished to get up and go down stairs; but he was in a state of extreme prostration, and evidently sinking. The pulse was 120 at the elbows, and imperceptible at the wrists. The sickness had ceased, but the features were pinched, and the skin was cold and covered with clammy sweat. He gradually sank, and died at 8 P.M.

Autopsy.—The abdomen only was examined. On opening this cavity the intestines appeared healthy, but distended with gas. There was no exudation or increased vascularity in the general cavity of the peritoneum. The large intestines contained a quantity of pulpy material of the colour of cream, and without any tinge of bile. The small intestines contained bile.

The left lobe of the liver was healthy and non-adherent. Both

the upper and under surfaces of the right lobe were connected to the adjoining parts by firm adhesions. Near the right edge of the liver a few of the bands of adhesion fastening it to the ribs appeared to be ruptured, and at this point there was a patch of recent lymph not larger than a square inch, with slightly increased vascularity round about. In the substance of the right lobe was an irregularly-shaped, collapsed cavity, the size of a large orange. The walls of this cavity were partly formed by the ribs and the surrounding adhesions. The inner surface of the cavity consisted of indurated hepatic tissue, presenting a shreddy appearance, and was not lined by any hydatid membrane. The cavity was almost empty: but it contained four or five collapsed hydatid vesicles about the size of a shilling. Communicating with it was a greatly dilated bile-duct, passing directly on to the common duct. The entire duct, from the cavity to the orifice in the duodenum, was large enough to admit the tip of the little finger. Further back in the right lobe, and quite distinct from the cavity now described, was another, about the size of a plum, which was lined by an obsolete and cribriform hydatid cyst, presenting a tough, opaque yellow appearance. The contents of this cavity had escaped during the hurried division of the liver. (This tumour was probably the source of the symptoms from which the patient had suffered seven years before his death.)

Case XI. appeared to be an example of an hydatid tumour of the liver, which had suppurated and become converted into an abscess in consequence of the entrance of bile. Considering the patient's condition before the operation, it is not probable that this contributed in any way to the fatal result.

CASE XI.—*Hydatid Cyst of Liver—Entrance of Bile—Inflammation—Paracentesis—Death.*

This patient was under the care of Mr. Moreton, of Tarvin, and Dr. Dobie, of Chester, by whom the fluid removed from the cyst was sent to me for examination.

Joseph B——, aged 58, a publican and huckster, had led rather an intemperate life. For three years he had often suffered from

attacks of vomiting and pain in the stomach, but he never had jaundice. On October 29, 1864, he was seized suddenly with severe pain in the stomach, greatly increased at intervals, and accompanied by tenderness on pressure, and incessant vomiting of glairy mucus. Calomel and opium were given in full doses, and warm fomentations applied. Next day the symptoms were much relieved, and for a fortnight the patient seemed to be improving. At the end of this time, the pain and tenderness of the abdomen had almost left him, and the vomiting had quite ceased; but the tongue remained coated, the appetite was bad, and the motions unnatural. On November 14, hiccup set in, which gradually became incessant, and about the same time the left lobe of the liver was noticed to become gradually enlarged. Ten days later there was unmistakable fluctuation over a space three inches in diameter, situated in the median line, two inches above the umbilicus. The man's condition became worse; incessant vomiting was substituted for the hiccup, and aphthæ formed on the tongue and cheeks, but there was no jaundice, and the motions always contained bile. On November 23, he had some shivering sensations, but at no time distinct rigors.

On December 1, a small trocar was introduced in the median line, two inches above the umbilicus, and about $1\frac{1}{2}$ pint of fluid was drawn off; and on December 6, a still larger quantity was removed. The patient, however, experienced no relief. He gradually sank, and died on December 10.

The fluid removed on December 1, after standing, consisted of (1) a clear, slightly yellowish, supernatant liquid, containing a mere trace of albumen, but a large quantity of chloride of sodium; and (2) of a copious yellowish-brown sediment, containing cholesterine and bile-pigment, but no pus.

The fluid removed on December 6 was of the consistence of thin pus, and of the colour of turmeric; and it contained numerous pus-corpuscles. No deposit or change occurred on standing.

No echinococci or hooklets could be discovered in either specimen.

No post-mortem examination of the body was permitted; but notwithstanding the absence of echinococci, the characters of the fluid removed on December 1 left little doubt in my mind that the tumour was an hydatid, which had become inflamed from the entrance of bile.

In Case XII. the hydatid tumour not only suppurated, but induced pyæmia, with secondary deposits of pus throughout the liver.

CASE XII.—*Suppurating Hydatid Tumour of Liver—Pyæmia, with Secondary Deposits of Pus.*

Thomas B—, aged 35, was admitted into the London Fever Hospital on January 20, 1866. He had lived for twenty years in Tasmania; but for the last four years in England. His previous health had always been good. His illness commenced five weeks before admission with severe pain in the right side, followed three weeks later by jaundice and diarrhœa. When seized with the pain, he first noticed a swelling in the right side; but this was as large then as at the time of admission. The patient was emaciated and jaundiced, and the liver was much enlarged, the vertical dulness in the right mammary line being eight inches. The portion of the liver projecting below the right ribs was smooth, painless, elastic, and almost fluctuating, but yielded nothing like ‘hydatid vibration.’ There was a moderate amount of ascites. Pulse 96; tongue moist and red; no appetite; six or seven liquid stools daily, containing little or no bile. Considerable sweating at night. Three or four days after admission, irregular attacks of rigors set in; the diarrhœa continued; the emaciation and perspirations increased; the tongue became dry and brown; and on February 22, the patient died. On two occasions (January 31 and February 7) an exploratory puncture was made into the tumour. On the first occasion nothing came away, owing to the trocar being too short; on the second occasion about six ounces of thin purulent bilious fluid was drawn off, which, unfortunately, was not submitted to microscopic examination. No bad consequence appeared to follow either operation.

At the autopsy an hydatid cyst, as large as a child’s head, and full of pus and secondary hydatids, was found projecting from the under surface of the liver, and compressing the portal vein and bile-ducts. The liver was studded with numerous small abscesses, and its outer surface was coated with recent lymph. Traces of the punctures were discovered with difficulty, and there was no evidence of increased inflammatory action in their neighbourhood.

In the following case the suppuration of an hydatid appears to have induced pyæmia, with secondary gangrenous abscesses in the liver. The anatomical characters of the liver agreed with those of ‘Gangrene of the Liver’ as described by Rokitansky.* The disease, however, is so rare that experienced observers have denied its occurrence, and Frerichs makes no mention of it. Even Rokitansky had met with only one example, and there it was associated with pulmonary gangrene. Budd reports one case, and quotes another from Andral.† Considering the rarity of such cases, the remarkably fetid odour observed during life is of considerable clinical interest.

CASE XIII.—*Suppurating Hydatid—Pyæmia, with Secondary Gangrenous Abscesses in the Liver.*

A man, aged 27, was admitted into the London Fever Hospital under my care on February 23, 1867. He was so prostrate that he could give little account of himself, and all that could be ascertained was that he had been a soldier in the West Indies for about seven years, but that his health had been good until about a month before admission, when he was seized with pain in the epigastrium and right hypochondrium, with nausea and vomiting, and about the same time he first noticed a tumour below the right ribs, the pain in which made it difficult for him to button his tunic over it. On admission he lay on his back, with his legs drawn up; the abdomen was full and tender all over; friction could be heard distinctly over the liver, which appeared large, extending downwards to the crest of the ilium, and upwards to the lower border of the third rib. The tongue was dry and brown, and there was frequent vomiting; but there was no jaundice, and the bowels were stated to be regular. The splenic dulness was increased; the pulse

* Path. Anat. Syd. Soc. Trans. vol. ii. p. 136.

† Budd, Op. cit. 3rd ed. p. 129.

was 132 and feeble; the heart's sounds were normal; the respirations were quick and thoracic; there was dulness on percussion over the back of the right lung, and moist sounds were heard over the greater part of both lungs. The skin was hot, the face pale, and the features pinched.

On the following morning the prostration had increased, and, in addition, there was noted slight jaundice of the conjunctivæ, and a peculiar, very fetid odour—*sui generis*, which appeared to proceed from the entire body, and not from the breath in particular. This was noted in the case-book before the patient's death, which took place on the same day.

On post-mortem examination, which was made on the day after death, there was found to be considerable evidence of recent peritonitis, particularly in the neighbourhood of the liver. Projecting from the under surface of the right lobe of the liver, and but slightly embedded in it, was an hydatid cyst, larger than a cocoa-nut. The wall of the parasite was opaque, tough, and cribriform, from the presence of numerous large openings, and its interior was filled with dirty brown purulent fluid, having a very offensive odour. The entire liver was studded with numerous softened masses from the size of a nut up to that of a small orange, in which the hepatic tissue was softened, and consisted of a spongy material, corresponding to the fibrous stroma and vessels, saturated with a greenish, extremely fetid pulpy fluid. Embedded in the substance of the liver, near the anterior edge of the right lobe, was a healthy hydatid cyst, about the size of a chestnut, containing clear fluid and echinococci. The lungs were congested, but were nowhere inflamed or gangrenous.

In the following case the hydatid tumour was so large as to almost fill the abdominal cavity, and bile had entered the cyst. The real nature of the case was not recognized during the patient's life, and paracentesis was resorted to merely as a palliative to relieve the patient's extreme distress, and with no idea of effecting a cure.

CASE XIV.—*Enormous Hydatid Cyst of the Liver, passing down through the Foramen of Winslow, and filling almost the whole of the Abdominal Cavity—Paracentesis—Pleurisy—Tubercle of Lungs—Death from Exhaustion.*

Elizabeth C——, aged 15, was admitted into the Middlesex Hospital, under Dr. Greenhow, August 26, 1862. She had been a very healthy infant, but at the age of 3 she had a severe fall on her right side, and since then she had never been well. For nine or ten years a swelling had been observed in the right side of the abdomen. Three years before she had been a patient in a London Hospital, but she had left on account of some operation having been proposed. The tumour increased gradually in size without causing pain, while at the same time the patient herself became thin and weak. Four weeks before admission she had been attacked with scarlatina, and during convalescence or for the last few days before admission, a very rapid increase had taken place in the size of the tumour, and there had been occasional pains in the abdomen. At the time of admission, the face and extremities were greatly emaciated; the countenance had a haggard, anxious expression, and the conjunctivæ were slightly tinged with yellow. The whole abdomen was enormously enlarged, and yielded distinct fluctuation; but the remarkable fact was that there was resonance on percussion in both flanks, as well as in the epigastric and both hypochondriac regions. The patient suffered from attacks of dyspnœa and of severe pain in the abdomen. Pulse 100, and feeble; no abnormal sound with heart; respirations hurried and thoracic; appetite good; bowels regular; urine very scanty and loaded with bile. On September 3, the abdominal pain and dyspnœa had become so distressing that the operation of paracentesis abdominis was performed as a palliative measure, and 248 ounces of a dirty brownish fluid were drawn off. The fluid was, unfortunately, not submitted to the microscope or to chemical reagents. The immediate effect of the operation was great relief to the pain and dyspnœa; but within three days the swelling was observed to be rapidly increasing, and on September 26 its dimensions were larger than before the operation, although the dyspnœa was not nearly so urgent. On the following day, the patient died from exhaustion.

Autopsy.—On dividing the abdominal parietes, about fourteen

pints of straw-coloured serum escaped. The greater part of the abdominal cavity, as far down as the pubes, was lined with a closely adherent gelatinous membrane, forming part of an enormous hydatid cyst, by which the stomach and intestines were pressed up closely against the under surface of the diaphragm and liver, where they were matted together, their peritoneal surface being considerably injected. Floating in the fluid, in the large abdominal cyst, was a secondary cyst containing about a pint of fluid and what appeared to be the débris of other cysts. Several cysts of smaller size were likewise found in the cavity of the large sac. On tracing the large primary cyst, it was seen to be continuous with a cyst about the size of a child's head projecting from, and attached to, the under surface of the liver. The two cavities, in fact, constituted one cyst, with an hour-glass constriction, the channel of communication being large enough to admit three fingers, and apparently corresponding to the foramen of Winslow. The gall-bladder was compressed, empty, and atrophied. Attached to the anterior border of the left lobe of the liver, by a thin fibrous peduncle, was another tumour about the size of a goose's egg, which, on being opened, was found to contain a crumpled-up hydatid cyst, filled with a putty-like material, in which were numerous hooklets of echinococci. A third tumour was found attached to the upper surface of the right lobe of the liver, and firmly adherent to the under surface of the diaphragm, which was pressed up into the cavity of the right pleura. This tumour was lined with a cyst, containing about a pint of straw-coloured serum, and the inner surface of which was studded with echinococci. The right pleural cavity contained about a pint of semi-purulent fluid, and the opposed surfaces of the pleura, at the base of the right lung, were coated with a deposit of recent semi-organized lymph. Both pleural cavities were much diminished in calibre by the elevation of the diaphragm, and both lungs contained a considerable amount of scattered miliary tubercles. The heart was small, but, in other respects, normal. The spleen was pale and shrunken. The kidneys were large and congested.

In the next three cases an hydatid of the liver proved fatal by opening into the pleura or lung.

The first case, which occurred while I was pathologist to the hospital, illustrates the absence of all

symptoms in a large hydatid tumour of the liver prior to its bursting into the pleura, and also the difficulty in diagnosis likely to arise from the co-existence of empyema with hydatid enlargement of the liver.

CASE XV.—*Hydatid Tumour of the Liver, bursting into the Right Pleura—Empyema—Death.*

Louisa R——, aged 17, was admitted into the Middlesex Hospital, under Dr. H. Thompson, March 23, 1861. She was a servant, and until a fortnight before she had continued at her work, enjoying good health, and not suffering any pain or uneasiness. She was then suddenly seized with acute pain in the upper part of the abdomen and on both sides of the chest, which was increased by inspiration, and was accompanied by cough, dyspnoea, febrile symptoms, and great prostration. On admission, pulse 112, small and weak. Slight cough. Dulness and absence of breathing over whole of right side of chest, except in infra-clavicular space. There was likewise dulness, with feeble breathing, at the base of the left lung. The hepatic dulness in the right mammary line extended nearly four inches below the margin of the ribs. There was no jaundice and no ascites, but the urine contained albumen. Hectic fever, with great prostration, set in, and death occurred on April 8, one month after the first symptom of illness.

Autopsy.—The heart was normal. The left lung was firmly and universally adherent: its lower lobe was hyperæmic, and near the base its tissue sank in water; but it was not granular on section, and it was unusually firm and tenacious. The right pleural cavity was filled with pus, floating in which were innumerable hydatid vesicles, from the size of a pin's head to that of an orange. The right lung was completely collapsed and carnified, except at the apex, which contained a little air. The liver was much depressed, its lower margin reaching to more than half-way between the umbilicus and the pubes. Projecting from the posterior margin of the right lobe was a cyst, as large as a child's head, which was firmly connected to the diaphragm; the liver was not adherent at any other part of its surface. At the upper part of the cyst there was a rupture through the diaphragm, measuring one inch and a-

half in diameter, by which the cyst communicated with the right pleura. The interior of the cyst was lined with an hydatid membrane; its cavity was filled with pus and vesicles. A large number of the vesicles were examined with the microscope, but no echinococcus or hooklet could be discovered. There was no other hydatid tumour either in the liver or in any organ of the body. The pelvis and calices of the right kidney and the upper part of the right ureter were dilated, apparently owing to the pressure below of the displaced liver; the secreting tissue of the right kidney was much atrophied; the left kidney was normal.

CASE XVI.—*Hydatid Tumour of the Liver, opening into the Right Pleura—Empyema—Pericarditis.*

George K——, aged 54, a gardener, of sober habits, was admitted into the Middlesex Hospital, under Dr. F. Hawkins, April 25, 1854. He had always enjoyed good health until four months before admission, when he was suddenly seized with pain all over the abdomen, but particularly in the right hypochondrium, and extending thence to the right shoulder. About the same time, he became slightly jaundiced. The pain and jaundice continued; and at the time the patient came under observation, he was very weak and emaciated, and suffered from incessant cough. The liver was much enlarged, extending down to the umbilicus. There was considerable bulging of the right side of the chest, which was universally dull on percussion, and devoid of respiratory murmur, except at the upper and back part close to the spine. The patient gradually sank, and died on May 10.

Autopsy.—On removing the sternum, the right pleural cavity was found to be filled with a yellowish, turbid, semi-purulent fluid, containing masses of a gelatinous substance, which proved to be hydatid cysts. The right lung was compressed and flattened against the vertebral column, and at its base was firmly bound by adhesions to the diaphragm. It did not crepitate in the least; it sank in water, and was completely carnified. The liver was enormously enlarged, extending downwards as far as the umbilicus, and weighing 90 ounces. It was firmly adherent to the diaphragm. In the posterior part of the right lobe was a cavity as large as a swan's egg, lined with an hydatid cyst, and containing similar cysts in its interior. The upper wall of this cavity was formed by

the diaphragm, and here there was a large opening by which the cavity in the liver communicated with the right pleura. The liver was much congested. The pericardium was glued to the heart by recent soft adhesions. The left lung, spleen, and kidneys were healthy.

CASE XVII.—*Old Hydatid (?)—Tumour of the Liver, communicating with Base of Right Lung—Lobular Pneumonia and Gangrene of the Lung.*

Robert J——, aged 72, was sent to the London Fever Hospital, August 21, 1864, as a case of ‘fever.’ On examination, he was found not to be suffering from any form of idiopathic fever. The man stated that he had had a bad cough for two months, and had kept his bed for two days. His breath had a most decidedly gangrenous odour. His sputa were of a dirty greenish mucopurulent character, and extremely fetid. Dry bronchial râles were audible over the chest, and at the right base there was slight dulness, with increased vocal resonance, and large moist râles, but nothing approaching to cavernous breathing. Pulse 96; respirations 36. No change took place in the physical signs of the chest; but the tongue became dry and brown, diarrhœa supervened, and the patient gradually lost flesh and strength until death on September 11.

On post-mortem examination, there was found to be lobular pneumonia of the lower lobe of the right lung, and quite at the base there was a gangrenous portion about the size of an orange. The lung was here firmly adherent to the diaphragm, and the diaphragm to the liver, and the broken-down tissue of the gangrenous lung communicated by several openings with a cavity in the upper part of the right lobe of the liver, measuring about three inches in diameter. This cavity contained much calcareous matter, and a quantity of a dirty greyish, very fetid, pultaceous substance. On careful examination, no hooklets of echinococci could be discovered. The rest of the liver and the intestines were healthy.

The absence of hooklets may be thought to negative the opinion that the tumour of the liver was originally an hydatid. But though these hooklets resist the changes which occur in the interior of the body for an indefinite period, they do not resist the putrefactive changes resulting from exposure to atmospheric air,

and such exposure must have existed here for many weeks before death. An obsolete abscess is the only other lesion that could have produced the appearances described, but the man had never suffered from the symptoms of abscess of the liver.

In the two following cases (and also in Case XV.) the tumour appeared to compress the ureters.

CASE XVIII.—*Hydatid Tumour of the Liver—Pyelitis—Pus in the Urine—Sudden Death.*

Ellen C——, aged 21, came under my care as an out-patient at the Middlesex Hospital, in April 1861. She stated that for about eighteen months she had been getting very weak and losing flesh, and that latterly she had suffered from dyspnœa. She had no cough, but her father had died of consumption. She had also suffered from irregular menstruation and leucorrhœa. On examining the chest, there was found to be a bulging of the right side, commencing at the upper border of the fifth rib, attaining its maximum at the false ribs, and then as gradually declining. The hepatic dulness in the right mammary line extended for 3 inches below the margin of the ribs, and its total length was $6\frac{1}{2}$ inches. The bulging below the ribs occupied the right hypochondrium and epigastrium, and extended over to the left hypochondrium. It was slightly tender, and presented an elastic, almost fluctuating consistence, and on percussion communicated to the finger the peculiar sensation known as ‘hydatid vibration.’ These characters were most marked in the epigastrium. The superficial veins about the epigastrium and hypochondrium were much enlarged. The movements of respiration were mainly confined to the left side of the chest. On the right side, the respiratory murmur could not be heard below the fourth rib in front, or below the lower angle of the scapula posteriorly. Above this the breathing was harsh, and the expiration was prolonged. On the left side there were also dulness and absence of respiration up to within half an inch of the lower angle of the scapula. The patient could give no information as to the length of time the tumour had existed. In fact, she was quite ignorant of the existence of any unusual swelling until it was pointed out to her. Her complexion was slightly sallow, but she had never suffered from jaundice or vomiting, and her bowels

were regular; her appetite was very bad. In addition to the tumour on the right side, a painful swelling, apparently an extension of the left lobe of the liver, could be felt in the left lumbar region in the situation of the kidney, and there was a copious discharge of pus in the urine.

The patient remained under my observation for nearly twelve months. The dimensions of the tumour did not alter much, but, on the whole, they became slightly larger. From time to time she suffered severe pain in the swelling in the left lumbar region. At these times the urine was clear, or almost so, and relief was always attended with a sensation of bursting and a return of the pus in large quantity. The urine was repeatedly examined with the microscope, but no pus-casts or trace of echinococci could be discovered.

The treatment—which consisted in the administration of tonics and iodide of potassium, and the external application of iodine—failing to give relief, the patient was admitted into the hospital on January 14, 1862, with the object of having a puncture made into the tumour in the right hypochondrium. After remaining in the hospital for six weeks she refused to give her consent, and was discharged at her own request.

I did not see the patient after this: but I have ascertained that, on November 6, 1863, she was admitted into University College Hospital, under the care of Dr. Hare, to whom I am indebted for the particulars noted while she was under his observation. Towards the end of 1862, she had first suffered from pain in the region of the tumour in the right hypochondrium. The pain was intermittent in its character, ceasing after a few days. For this she had been treated at the Female Hospital in Soho Square. The dimensions of the tumour noted in University College Hospital showed that it had increased considerably. Although the right costal angle was still greater than the left, there was bulging of the ribs on both sides as high as the nipple, and dulness on percussion up to the third rib on the right side, and up to the third intercostal space on the left side. The heart was displaced upwards, its apex beating in the third left intercostal space. The vertical hepatic dulness in a line with right nipple was $11\frac{1}{4}$ in.; in the median line, $9\frac{1}{2}$ in.; and in a line with left nipple, $9\frac{3}{4}$ in. Distinct fluctuation could be felt in the epigastrium over a space measuring $4\frac{1}{2}$ in. transversely, and $2\frac{1}{2}$ in. vertically; but there was now no hydatid fremitus. There was no œdema of the legs. The

patient was sallow; her urine contained no bile-pigment, but was still loaded with pus. She still suffered from the attacks of pain in the region of the left kidney, which were always relieved by a sensation of bursting and a copious discharge of pus in the urine. On admission, there was a considerable amount of pain and tenderness in the region of the tumour near the umbilicus. This pain recurred from time to time, but was always relieved by leeches, poultices, and morphia. The patient also had an attack of pain and stiffness in the left groin and knee, accompanied by enlargement of the lymphatic glands in the groin, and slight oedema in the upper part of the thigh. On January 26, 1864, it was noted that she was free from pain, but that she had lost flesh and strength. On February 9, she was discharged for unruly conduct.

The patient was confined to bed after leaving the hospital, and died rather suddenly and unexpectedly at the end of ten days. An hour before death she seemed tolerably well, and the probability is that the fatal event was due to the bursting of an hydatid cyst.

CASE XIX.—*Hydatid Tumours of the Liver and Peritoneum, compressing the Ureters, and causing Degeneration of the Kidneys.*

Mary Ann W——, aged 45, was admitted into the Middlesex Hospital, December 15, 1864, under the care of Dr. H. Thompson, and died January 15, 1865. For a year before death she had suffered from headache and impairment of the mental faculties, and seven weeks before death she had a fit of unconsciousness, followed by right hemiplegia, involuntary evacuations, and bed-sores. There were no symptoms referable to liver.

The arteries at the base of the brain were atheromatous, and there was an apoplectic cyst, with a patch of white softening in the left corpus striatum. The liver, spleen, and diaphragm were adherent by fibrous bands. In the adhesions between the spleen and liver was a cyst the size of a walnut, filled with soft putty-like matter, and lined by portions of a gelatinous echinococcus membrane. In the right lobe of the liver was another cyst, the size of a small cocoa-nut, partly embedded in its substance, and partly projecting from its upper surface, where it was firmly ad-

herent to the diaphragm. Its outer wall was partly calcified, and its anterior was full of fragments of secondary gelatinous cysts, and soft, putty-like matter. The secreting tissue of the liver was healthy. In the folds of the mesentery of the small intestine were three partly calcified cysts, varying in size from a hazel-nut to a walnut, and containing putty-like matter and secondary cysts. The greater part of the pelvis was occupied by another large cyst, situated behind and above the uterus, which was forced down, so as to appear at the vulva. This cyst contained a clear fluid and innumerable small cysts, varying in size from a pea to a walnut, all of them gelatinous and filled with a clear fluid. There was another cyst, not so large, in the right side of the pelvis. The ureters were compressed by these cysts, and the pelves of the kidneys were somewhat dilated. The kidneys were small and granular, and the cortices were wasted and hardly distinguishable from the cones. All of the cysts in the abdomen contained numerous teeth of echinococci.

In the following case secondary hydatid cysts were formed in the omentum and peritoneal cavity.

CASE XX.—*Hydatid Cysts of the Liver and Peritoneum—Ascites and Anasarca of Lower Extremities—Albuminuria—Death.*

Catherine C——, a hawker, aged 45, was a patient in the Middlesex Hospital, under Dr. Goodfellow, from January 10, 1865, until her death on June 21. With the exception of an attack of rheumatism, she had enjoyed good health until about a month before admission, when she had been seized with violent pain in the abdomen and loins, and at the same time her legs and abdomen had begun to swell. While in hospital, she suffered from ascites and great anasarca of the lower extremities. The urine contained albumen. She was treated with diuretics and purgatives, and her legs were punctured.

On examination of the body, the legs were observed to be very cedematous, and the abdomen was greatly distended. Both lungs were very cedematous, and the right lung was firmly adherent and carnified at its base.

The peritoneal cavity contained upwards of a gallon of clear serum, floating about in which were six nearly transparent hydatid cysts, with tremulous gelatinous walls, the largest about the size of a hen's egg, and the smallest about that of a walnut. The fluid in the floating cysts had a specific gravity of 1010, and contained no albumen; that in the peritoneal cavity had a specific gravity of 1020, and was highly albuminous. The left lobe of the liver was partly atrophied, and between it and the spleen, and firmly adherent to both and to the stomach was an hydatid cyst, the size of a foetal head, containing a little clear fluid and innumerable smaller cyst of various sizes pressed together. In the great omentum were three or four similar cysts the size of chestnuts, and attached to the right kidney was another cysts as big as an orange. Numerous echinococci were found in the larger cysts. Both kidneys were much enlarged and fatty.

CASE XXI.—*Hydatid Tumour of the Liver—Secondary Hydatid Tumours in the Spinal Canal—Paraplegia.*

The preparation of this case is in the Museum of Middlesex Hospital (v. 15), and the following particulars are extracted from the Catalogue :—

‘Vertebræ with spinal cord from dorsal region. The canal and dura-mater laid open. The pleura is separated from the ribs and the sides of the bodies of the vertebræ by two hydatid cysts, one on each side. The hydatids have been opened in sawing through the laminae of the vertebræ; but their walls remain, and the spinal cord is at this place considerably smaller than elsewhere.

‘The patient was a women aged 40, who had been admitted into the hospital with paraplegia and retention of urine. She died with a large slough on the sacrum, and the bladder was found to be inflamed. There was also a large hydatid cyst in the liver.’

In the following case a process of spontaneous cure appears to have commenced in the tumour, and the observation is interesting in connection with the manner in which a cure is probably effected in an

hydatid tumour, when the fluid contents are drawn off by means of a small trocar and canula (see pages 62 and 76).

CASE XXII.—*Large Hydatid Tumour of the Liver, full of Secondary Cysts, but containing no Fluid.*

This liver was taken from the body of a man, aged 36, who was admitted into the Fever Hospital on December 2, 1866, with petechial small-pox, of which he died on December 5. He was too ill to give any particulars of his previous history.

After death, an hydatid tumour, the size of a child's head, was found in the posterior part of the right lobe of the liver. The chief point of interest in the case was that this cyst was tightly packed with secondary cysts, but that it contained no fluid. The secondary cysts were collapsed; but still they exhibited their natural gelatinous appearance. They were not at all opaque or mixed up with any putty-like material. The outer cyst, however, at several places presented an atheromatous calcified appearance.

Note.—The practical conclusion to be deduced from the annexed Table (p. 118) is identical with that published by me in the 'Edinburgh Medical Journal' for December 1865, but is opposed to that arrived at from a similar enquiry by Dr. John Harley, and published in the 49th volume of the 'Medico-Chirurgical Transactions' (1866). Dr. Harley, who advocates the treatment of hydatid tumours of the liver by a large and permanent opening, gives a Table of 34 cases which were treated by a single puncture, evacuation of a portion or of the whole of the fluid, and immediate closure of the wound,' and states that, 'there were 11 cures, 13 recoveries, i.e. cases which were relieved by the operation, but which, since the tumour was not wholly removed, or the result sufficiently certified, cannot be regarded as radical cures, and 10 deaths.' Inasmuch as the parent and secondary cysts can never be 'wholly removed' by the operation of simple puncture, it is difficult to understand how Dr. Harley can admit that there was a 'radical cure' in any of the 34 cases. It is necessary, therefore, to explain that he seems to look upon the result as a *recovery*, and not a *cure*, if any trace of the tumour can be *felt* some time after the operation (as in my own case, No. 25 in his Table). The introduction of the 10 fatal cases into

the Table, however, throws, in my opinion, an illegitimate discredit upon the operation in question, and it is, therefore, necessary to advert to them in detail.

Case 4.—In this case the tumour filled up the whole abdomen, and the operation of paracentesis (with a large trocar) was resorted to, with the object of relieving the impending asphyxia, and not as a curative measure. The patient, moreover, before the operation, was in a state of extreme marasmus and prostration, and the immediate cause of death was miliary tubercles in the lungs, and empyema. See Greenhow, 'Lancet,' 1862, ii. p. 476, and Murchison, 'Ed. Med. Journ.' Dec. 1865.

Case 8.—There is no evidence that this case was fatal. Dr. Harley quotes the case from Mr. Cæsar Hawkins, and Mr. Hawkins from Dr. Thomas's 'Practice of Physic.' Mr. Hawkins observes, 'The result is not mentioned, so that it may probably be concluded that the case ended fatally,' but Dr. Thomas says nothing to warrant such a conclusion. 'Med.-Chir. Trans.' vol. xviii. p. 121.

Case 9.—The operation was resorted to merely as a palliative measure: 8 pints of fluid were withdrawn from one cyst, and a second cyst, containing 12 pints, was found after death between the liver and the diaphragm. Dr. Abercrombie adds, 'The two cysts had so much injured the patient's constitution, that, although he was relieved by the operation, his strength quickly failed him.' Abercrombie, 'Dis. of Stomach,' p. 356.

Case 10.—In this case the opening was evidently a large one, and it is not stated whether it was closed up or not. But what is more important, the hydatid had supplicated before the operation. Hawkins in 'Med.-Chir. Trans.' vol. xviii. p. 157.

Case 11.—From the original account of this case in the 'Edin. Essays and Observ.' vol. ii. p. 299, it is clear that the boy was almost moribund at the time of the operation, and that, in addition to hydatids of the liver and spleen, he had ascites, general dropsy, and orthopnoea. It seems probable also that the peritoneum, and not the hydatid, was tapped.

Case 13.—In this case there was great constitutional disturbance, and the hydatid had supplicated before the operation. The patient also was pregnant and miscarried, and sank after this. Dr. Bright on 'Abdom. Tumours,' Syd. Soc. Ed. p. 41.

Case 15.—In this case there were two hydatid tumours. Three pints of fluid were drawn from one. This cyst did not again become enlarged, and the patient fancied herself cured, when

death occurred from the rupture of the other cyst through the diaphragm into the lungs. Davaine, '*Traité des Entozoaires*,' p. 447.

Case 16.—In this case the patient was in a state of extreme prostration before the operation. He was seized with syncope within five minutes, and died at the end of eighteen hours. Traces of recent peritonitis were found after death. The fatal result was no doubt determined in this case by the operation, but a large opening left patent is not likely to have led to a more favourable termination. *Archiv. Gén. de Méd. sér. v. tom. xiii. p. 145.*

Case 19.—In this case the puncture was simply an exploratory one, preparatory to the application of caustic potash seven days afterwards. Death was due to tetanus twenty-five days after the puncture, and Récamier states, '*aucun accident n'a suivi la ponction.*' Davaine, *Op. cit.* p. 590.

Case 32.—In this case, according to Dr. Harley, no attempt was made to relieve the sac of its contents after the first puncture, and the hydatid fluid escaping into the peritoneum caused peritonitis and extension of the disease; but he omits to mention that the presence of a large and increasing amount of fluid in the peritoneum was diagnosed before the operation. Moreover, caustic potash was applied to the integuments before the cyst was tapped. Rogers in '*Brit. Med. Journ. 1862*,' vol. i. p. 71.

Since the above lecture was delivered the following case has come under my notice,* which is an illustration of another disease that may be confounded with hydatid tumour of the liver. The tumour during life was believed to be an hydatid, with which the history of an injury is in no way incompatible, for in many cases of hydatid the patients date their origin from an injury, which probably acts merely by attracting attention to a tumour already existing. I

* Similar cases are recorded by Mr. Cæsar Hawkins in the 18th volume of the *Med.-Chir. Transactions*, p. 175; by Mr. Stanley in the 27th volume, p. 1; and by Sir Henry Thompson in the 13th volume of the *Path. Transactions*, p. 128.

know no rule by which a similar mistake might be avoided in future, but in most cases of cystic tumours connected with the urinary passages and arising from injury there would probably be: 1. A history of hæmaturia or albuminuria, or some symptoms of urinary irritation, all of which were absent in this case; and 2. Urea would be found in the fluid drawn off by tapping. Unfortunately, the fluid drawn off in this case was not examined for urea, but none was found in that which remained in the sac after death. The operation was resorted to merely as a palliative, and contributed in no way to the fatal result; the inflammation of the sac and the secondary deposits in the lungs had commenced previously.

CASE XXIII.—*Enormous Cystic Tumour communicating with the Pelvis of the Right Kidney, existing for Eight Years, and simulating Hydatid Tumour of the Liver.*

Joseph O——, aged 16, was admitted into the Middlesex Hospital under my care on December 19, 1867. Eight years before he had been thrown with great force against a wall, injuring his back and right side. For a week he vomited everything he swallowed, and altogether he was laid up for two months, but he never was observed to pass blood in his urine, or to have urinary symptoms of any sort.

He then went to school for a month, when he was seized with severe pain in his back and right side, for which leeches were applied. He was in bed for five months, and during this time he had frequent vomiting and nine fits of convulsions, the movements being limited to the left side of the body. Shortly after this his mother noticed that his right side had ‘grown out,’ and the swelling increasing she took him to the London Hospital, where he remained for four months, and where his general health

underwent great improvement. His health continued good, and he was able to go about, but the swelling went on slowly increasing, until about a week before admission, when, after getting thoroughly wet outside a cab, he was seized with severe pain in the back, cough, and febrile symptoms.

On admission, the patient was anæmic and emaciated, and complained of cough and shortness of breath, and of great pain and tenderness in the lower part of the spine. Pulse 108; respirations 48 and thoracic; bronchitic râles over whole of both lungs, with dulness and friction over lower fourth of left. Tongue clean; appetite bad; temperature $101^{\circ} 4$. There was no anasarca, and the urine contained no albumen. But the most remarkable feature about the boy was the enormous size of the abdomen, which measured $33\frac{1}{2}$ inches at the umbilicus, the bulging being greatest in the right flank. This enlargement was almost painless, and was evidently due to an encysted collection of thin fluid in the right side, extending from the liver down into the pelvis, and as far forwards as the middle line, but clearly shut off from the general cavity of the peritoneum, as the rest of the abdomen was tympanitic in whatever position the patient lay. The hepatic dulness ascended to the nipple in front, and to the lower angle of the scapula behind.

After admission the tumour increased in size, and the dyspnœa became so urgent that, on December 23, it was resolved to tap the cyst, which was accordingly done by Mr. Hulke, midway between the ribs and the crest of the ilium, and 170 ounces of fluid drawn off. The fluid which first came away was clear, but of a brownish colour; its specific gravity was 1010, and it contained much chlorides, and about one-sixth of albumen. The last two pints contained much pus, forming on standing a creamy deposit, of about one-half of the entire bulk. No portion of the fluid contained either echinococci or hooklets.

At first the operation was followed by great relief to the dyspnœa, and at no time afterwards had the patient either rigors, profuse perspirations, pain in the tumour, or albumen in the urine. The prostration, however, increased daily; the tongue became dry; the temperature varied from 100° to $103^{\circ} 2$; there was much restlessness with sleeplessness and occasional delirium, and the signs of pleurisy at the base of the left lung, noted before the operation, extended. He gradually sank, and died on January 2, 1868.

Autopsy.—There were no signs of recent peritonitis, but on the right side of the abdomen, lying behind the intestines, was a cyst, with thick fibrous walls, about the size of an adult human head, It was firmly attached by fibrous adhesions to the under surface of the liver, to the false ribs, and to the abdominal wall. It extended downwards to the brim of the pelvis, and slightly beyond the middle line to the left. The right kidney was expanded over its outer and posterior aspect, and the renal tissue was attenuated and wasted. On opening the sac, its contents amounted to 65 ounces of thin pus; its inner wall presented a fibrous puckered aspect, with no trace of hydatid structure, and it communicated by three openings, oblique and valvular, but large enough to admit a full-sized catheter, with the pelvis of the kidney.* The right ureter was rather small, but pervious throughout; it ran for some distance in the wall of the cyst immediately beneath its lining membrane, and then passed down to the bladder, which was quite normal. The upper part of the right kidney was converted into a cicatrix-like fibrous tissue, intimately incorporated with the cyst. The left kidney was double the normal size, but otherwise normal. The liver was fatty; the spleen was very large and soft. There was recent pleurisy over the lower lobe of the left lung, which contained a patch of red hepatization; and in the lower lobe of the right lung were several small patches of lobular pneumonia, with yellow centres. There was no pus in the joints, and no sign of old fracture of the ribs, or of disease of the bodies of the vertebræ.

* It is remarkable that, notwithstanding these openings the urine, up to the day of death, never contained any pus or a trace of albumen. A similar observation was made in the case recorded by Mr. Cæsar Hawkins, and already referred to (p. 114, *foot-note*). In that case also, although the cyst communicated with the pelvis of the right kidney, no urea could be found in the contained fluid, which is also said to have been devoid of albumen, although it contained pus!

Table of Cases of Hydatid Tumour of Liver treated by a Simple Puncture, and Closure of Opening after Evacuation of Fluid.

No.	Med. Attendant	Quantity of Fluid Withdrawn	Result	References and Remarks
1	Murchison	5 oz. and 20 oz.	Cure	<i>Antea</i> , Case VI. p. 83.
2	Do.	12 oz.	Cure	<i>Antea</i> , Case VIII. p. 85.
3	Do.	14 oz.	Cure	<i>Antea</i> , Case VIII. p. 89.
4	Greenhow	21 and 110 oz.	Cure	Path. Trans., vol. xviii. p. 127.
5	Do.	148 oz.	Cure	Ib. p. 130.
6	G. Budd	150 oz.	Cure	Med. Times and Gaz. May 19, 1860.
7	Holthouse	100 oz.	Cure	Ib. Jan. 6, 1855.
8	J. Hutchinson	30 oz.	Cure	Lancet, 1862, vol. ii. p. 389.
9	Sir B. Brodie	30 oz.	Cure	Med.-Chir. Trans. vol. xviii. p. 118.
10	Do.	60 oz.	Cure	Ib. p. 119.
11	Dr. Thompson	60 oz.	Cure	Ib. p. 121.
12	Récamier	Small quantity	Cure	Rév. Méd. Tom. i. p. 28.
13	Key	80 oz.	Cure	Bright on Abd. Tumrs. Syd. Soc. Ed. p. 42.
14	Boinet	20 oz.	Cure	Traitement des Tumeurs hyd. du Foie, Paris, 1859, p. 13.
15	Do.	4 oz.	Cure	Ib. p. 14.
16	J. Hutchinson	40 oz.	Cure	Brit. Med. Jour. Feb. 20, 1864.
17	Do.	60 oz.	Cure	Ib.
18	Frerichs	120 oz.	Cure	Dis. of Liver, Syd. Soc. Ed. vol. ii. p. 268.
19	Langenbeck	?	Cure	Ib.
20	Do.	?	Cure	Ib.
21	W. Budd	23 oz.	Cure	Brit. Med. Jour. 1859, p. 273.
22	Robert	?	Cure	Gaz. des Hôpitaux, 1857, p. 147.
23	Do.	?	Cure	Soc. de Chir., Mars 18, 1857.
24	Brinton	30 oz.	Cure	Lancet, 1862, vol. ii. p. 639.
25	Richard	40 oz.	Cure	Bull. Gén. de Therap. 1855, p. 414.
26	Demarquay	20 oz.	Cure	Boinet, Op. cit. p. 30.
27	Boinet	20 and 15 oz.	Cure	Ib. p. 18.
28	McGillivray	30 and 20 oz.	Cure	Austral. Med. Journ. Aug. 1865. Case iii.
29	Do.	?	Cure	Ib. Case vii.
30	Do.	180 and 100 oz.	Cure	Ib. Case xv.
31	Do.	114 oz.	Cure	Ib. March 1867. Case xxiv.
32	Do.	20 oz.	Cure	Ib. Case xxvi. Three distinct cysts were tapped, none of which refilled.
33	Do.	2 oz.	Cure	Ib. Case xxxv.
34	Do.	18 oz.	Cure	Ib. Case xxxvi.
35	Do.	70 oz.	Cure	Ib. Case xxxviii.
36	Owen Rees	38 oz.	Suppuration Free Ext. Open ^s Cure.	Guy's Hosp. Reports, ser. ii. vol. vi. p. 17.
37	Garrod	4 oz.	Do. do. do.	Lancet, Sept. 1, 1860.
38	Boinet	40 oz.	Do. do. do.	Gaz. Hebdom. de Méd. sér. ii. 1864, i. p. 80.
39	Demarquay	160 oz.	Do. do. do.	Gaz. des Hôp. Fév. 19, 1859, p. 82.
40	Babington and Cock	10 and 80 oz.	Do. do. do.	Guy's Hosp. Rep. 3rd. ser. vi. p. 179. In this case the object of the operation was not to remove all the fluid at once, but by repeated punctures.
41	McGillivray	20 oz.	Do. do. do.	Austral. Med. Jour. Aug. 1865. Case xiv.
42	Do.	10 oz.	Do. do. do.	Ib. March 1867. Case xix. The fluid was milky at first operation.
43	Do.	Large quantity	Do. do. do.	Ib. Case xxxiii.
44	Do.	60 oz.	Do. do. death	Ib. Case xvi. Death was due to the bursting of the tumour into the lung after the large external opening was made.
45	Wiltshire	Large quantity	Do. do. death	Lancet, Sept. 1, 1860. The liver contained three other cysts, each containing about a pint of fluid, besides the one that was punctured. Death was mainly attributed to the pressure of the liver on neighbouring organs.
46	Moissenet	12 oz.	Death	Arch. Gén. de Méd. Fév. 1859, p. 144. The patient was extremely prostrate before the operation, and died of peritonitis eighteen hours after.

LECTURE IV.

ENLARGEMENTS OF THE LIVER.

CONGESTION—INFLAMMATION OF BILE-DUCTS—OBSTRUCTION OF COMMON DUCT.

GENTLEMEN,—In the previous lectures I have called your attention to the distinguishing characters of the four enlargements of the liver which are for the most part unattended by pain. Those in which pain is a prominent symptom remain to be considered. Six diseases are included under this head; viz. 1, congestion of the liver; 2, inflammation of the bile-ducts; 3, obstruction of the common duct and retention of bile; 4, pyæmic abscesses; 5, tropical abscess; 6, cancer. Speaking generally, it may be said that jaundice, which is a rare symptom in painless enlargements of the liver, is present to a greater or less extent in the class of enlargements now to be noticed. Tropical abscess is the one in which it is oftenest absent. First among the enlargements of the liver attended by pain comes—

V. CONGESTION OF THE LIVER.

In the first place, it is necessary to bear in mind, in reference to the pathology and treatment of this

condition, that the quantity of blood in the liver varies greatly at different times consistently with health, and that even these healthy variations may influence to some extent the size of the organ. For instance, the amount of blood in the liver and its size are greatly influenced by diet, both being temporarily increased after a meal, and particularly when the food has been too large in quantity, or contained an excess of fatty, saccharine, or alcoholic ingredients. By morbid congestion of the liver, we mean something more than this. The phrase 'congestion of the liver' is too often used very vaguely and applied to cases of indigestion, where there is probably little amiss with the liver. True congestion of the liver is distinguished by the following characters:—

1. There is enlargement of the liver which is uniform in character—not greater in one direction than another—and which is rarely very great. The liver may project an inch or more below the margin of the ribs in the right mammary line. In the venous engorgement from mechanical obstruction of the circulation, the enlargement is usually greater than in active congestion, where the engorgement commences in the arteries. Another peculiarity of this enlargement is that it is rarely permanent, but that after a time it usually disappears. Even when the cause of the obstruction is most permanent, such as mechanical obstruction of the cardiac circulation from valvular disease of the heart, the primary enlargement of the liver from congestion gives place after a time to an

opposite condition of contraction. The pressure exerted by the constantly distended hepatic veins causes atrophy of the central portions of the lobules, and induces a form of granular liver, very different from true cirrhosis, where the atrophy commences at the circumference of the lobules. In true cirrhosis of the liver most observers have noted a temporary enlargement of the liver from congestion in the early stage of the malady;* and although this has been doubted by the late Dr. Todd† and others, there can be little doubt that at the commencement of cirrhosis there is congestion, with more or less enlargement, due to the excessive amount of alcoholic ingesta on which cirrhosis ordinarily depends. But here, as in other forms of congestion, the enlargement is of temporary duration.

2. The surface of the portion of liver projecting below the ribs is perfectly smooth.

3. The patient complains of a feeling of tightness or painful distension in the region of the liver, and there is more or less—but rarely very acute—tenderness on pressure below the margin of the right ribs. The pain and feeling of uneasiness may stretch up to the right shoulder, and are almost always increased after meals, or by lying on the left side. In the latter case, there is usually a sense of dragging or weight in the hepatic region. The patient consequently sleeps for the most part on his back, or on his right side.

* Frerichs : Klinik der Leberkrankheiten. Syd. Soc. Ed. ii. pp. 37, 53.

† Clinical Lectures on Urinary Diseases and Dropsies, 1857, p. 113.

4. Jaundice is present, in most cases, after two or three days, but is rarely intense, and it is not often that bile is altogether absent from the motions. When there is intense jaundice with absence of bile from the stools, catarrh of the ducts is probably present, as well as congestion of the hepatic tissue.

5. There is usually nausea, with loss of appetite, headache, furred tongue, flatulence, and other symptoms of indigestion, and not unfrequently there is vomiting or diarrhoea, or both. The same cause that produces congestion of the liver may induce a similar condition of the stomach and intestines. Slight irritation then suffices to induce catarrhal inflammation of the mucous membrane of these parts, of which vomiting and diarrhoea are the prominent symptoms.

But when the hepatic congestion is independent of mechanical obstruction of the circulation, the bowels are oftener constipated than relaxed, and the patient's chief symptoms are loss of appetite, furred tongue, a bitter taste in the mouth, nausea, and flatulence, with general languor and debility, anæmia, emaciation, and hypochondriasis.

6. More or less dyspnoea is not uncommon, even in cases where the primary disease is not in the chest, and many patients are harassed by a frequent dry cough.

7. Signs of obstructed circulation are not uncommon. In acute cases there may be tension in the left hypochondrium, and an increased area of

splenic dulness ; while in more protracted cases there may be hæmorrhoids or ascites.

8. The urine is usually scanty and high coloured, and besides containing more or less bile pigment, often deposits a copious sediment of lithates or lithic acid.

9. As in other forms of enlargement of the liver, the circumstances under which the enlargement appears constitute an important aid to the diagnosis of the real nature of the case. Hepatic congestion may be mechanical, active, or passive, and the chief conditions under which it occurs are the following :—

A. *Mechanical*.—Among the most common causes of hepatic congestion in this country is mechanical obstruction of the circulation in the chest, and particularly that consequent on disease of the mitral or tricuspid valves of the heart. In many cases of valvular disease of the heart, a time arrives when the chief symptoms are those of hepatic congestion, and the main treatment must be directed to their relief.

B. *Active*.—Several causes contribute to the development of active congestion :—

a. Irritating ingesta, in the form of alcohol, fermented liquors, spices, or food which errs in being habitually too rich in quality or in excessive quantity may cause congestion of the liver. The temporary increase of blood which the liver always contains after a meal may become morbid in degree and permanent, if the ingesta be habitually of an irritating character. Congestion of the liver is more

likely to result from these causes in weakly persons who lead indolent and sedentary lives, than in persons of a robust constitution who take plenty of muscular exercise in the open air.

b. A high temperature is usually reckoned among the causes of congestion of the liver, but probably rarely leads to such a result except in conjunction with irritating ingesta. It is to this combination of causes that must be attributed the frequency of active congestion of the liver among Europeans in warm climates.

c. A sudden or protracted chill in warm climates may increase the congestion arising from the causes now mentioned, and may even induce inflammation and abscess.

d. *Malaria and Blood-Poisons*.—Persons who suffer from malarious fevers, or live in malarious districts, are very prone to have congestion of the liver, which may persist long after the febrile symptoms have passed away. Officers and soldiers not uncommonly return from India with enlargement of the liver from this cause. But when great and permanent enlargement of the liver succeeds to ague or remittent fever, it is more probably the result of waxy deposit than of simple congestion.* There are other blood-poisons besides malaria, which may induce congestion of the liver, such as the yellow fever of the tropics, and the relapsing fever of our own country.

* See page 30 and Case II., and also Morehead, Res. on Dis. in India, 1860, p. 428; and Sir Ranald Martin, in Lancet, 1865, ii, p. 615.

e. Active congestion of the liver may have a traumatic origin, and result from contusions, wounds, &c.

C. Passive.—Passive congestion of the liver may be due to—

a. Suppression of habitual discharges, as of the catamenia or of the bleeding from piles.

b. Habitual constipation.*

c. Torpor of the portal vascular system from paralysis of the sympathetic nerves or from any other cause.

Treatment.—In the treatment of hepatic congestion, you must be guided by the following rules:—

1. In most cases of any severity advantage will be derived from the employment of local depletion in the form of leeches or cupping to the region of the liver, or, what is still better, the application of a few leeches around the anus. If depletion be deemed inexpedient, sinapisms may be applied over the liver. After the leeches or the sinapisms, their place ought to be supplied by linseed or bran poultices. Tepid baths are sometimes useful.

2. The diet should be of the least irritating character. Only small quantities of milk, beef-tea, or farinaceous articles ought to be taken at a time. Alcohol, wine, fermented liquors, spices, fat, and all rich or indigestible articles ought to be rigidly interdicted. In modern practice much mischief is often done by

* See Frerichs, Dis. of Liver, Syd. Soc. Transl. i. p. 376.

compelling patients with heart-disease and congestion of the liver to swallow large quantities of brandy.

3. Purgatives are in most cases of great utility, unless there be spontaneous diarrhoea, which ought not to be too speedily or completely checked. The best purgatives are those which increase the watery exhalation from the mucous membrane of the bowels, such as the sulphates of soda and magnesia, seidlitz powder, Püllna water, the citrate of magnesia, and the bitartrate of potash. The action of these purgatives is sometimes materially assisted by an occasional dose of calomel, blue pill, or podophyllin, which bring away copious bilious motions.*

4. When the congestion is traceable to irritating ingesta, an emetic in the early stage sometimes appears to do good, by clearing out the stomach and duodenum. The pressure also to which the liver is subjected during the act of vomiting may squeeze out of it some of the superfluous blood.

5. During the persistence of the symptoms of congestion—enlargement and tenderness of the liver with jaundice—and especially in those cases where there is much gastric derangement, alkalies and their salts with the vegetable acids ought to be prescribed. They may be taken two or three times a

* The increased biliary excretion after the calomel in these cases is not due to an increased secretion of bile by the liver, but probably to the mercury irritating the upper part of the small intestine, so that the bile is propelled onwards, instead of being re-absorbed (see Lect. VIII). If calomel acted by stimulating the liver to increased secretion, it would be injurious in cases of hepatic congestion.

day on an empty stomach. The alkaline mineral waters, such as those of Vals, Vichy, and Ems, or the artificial effervescing Vichy salt, may often be advantageously substituted for the alkaline preparations of the Pharmacopœia.

6. When the more urgent symptoms have passed off, and the patient chiefly suffers from debility and dyspepsia, with a slight increase of the hepatic dulness, with or without hypochondriasis, the treatment must be modified. The mineral acids and vegetable tonics are now most useful, such as the mineral acids with taraxacum or gentian, or small doses of quinine and iron. The latter remedies are particularly indicated in patients who have suffered from malarious fevers. The diet also ought to be more generous, although care must be taken to exclude from it every source of irritation. Fermented liquors ought still to be absolutely interdicted, and if wine be allowed at all, it should be given in small quantities and diluted. Dry sherry and claret are the best. Regular exercise in the open air ought to be enjoined ; if there be much debility, the advantages of exercise without fatigue may be derived from riding on horseback. The bowels will still require attention, and great advantage will often be obtained from the use of mineral waters which combine chalybeate with purgative properties, such as the springs of Harrogate, Cheltenham, and Leamington.

7. It is in the chronic condition last referred to that advantage is often derived from the use of the

nitro-muriatic acid bath, as recommended by Sir Ranald Martin.* The bath should consist of two ounces of strong hydrochloric, and one ounce of strong nitric acid to two gallons of water, at a temperature of 96° or 98°. Both feet are to be placed in the bath, while the inside of the legs and thighs, the right side over the liver, and the inside of both arms, are sponged alternately, or the abdomen may be swathed in flannel soaked in the fluid. The process is to be continued for half an hour night and morning.†

As an example of congestion of the liver resulting from mechanical obstruction of the circulation in the chest, I may recall your recollection to the following case:—

CASE XXIV. — *Mitral Constriction—Dropsy and Congestion of the Liver—Death.*

Emma F—, aged 13, was admitted under my care on October 24, 1865, suffering from much cough, great dyspnœa, and considerable anasarca of the lower extremities. The cardiac dulness had double its normal area, and a prolonged bellows-murmur was audible over the left apex. There were all the signs of general • bronchitis; and, in addition, the conjunctivæ and general surface had a slightly jaundiced tint; the hepatic dulness was much increased, measuring in the right mammary line, more than five inches, and extending down nearly to the umbilicus. The splenic dulness was also increased. There was considerable tenderness on

* See Lancet, Dec. 9, 1865, p. 641.

† The bath, as above prepared, may be kept in use for a few days, 1 drachm of hydrochloric, and half a drachm of nitric acid, with a pint of water, being added daily to make up for waste. About a fourth of the fluid is to be well heated in an earthen pipkin, so as to bring up the temperature of the whole to 96° or 98°. Glazed earthen or wooden vessels should be used, and the sponges and towels kept in cold water lest the acid corrode them.

pressure below the right ribs. The tongue was furred. There was much nausea and occasional vomiting, and the bowels were relaxed about four or five times a day. The motions were pale, though coloured with bile. The urine contained a small amount of bile-pigment, but no albumen. Five or six years before, this patient had an attack of scarlet fever, followed by articular rheumatism and dropsy. Ever since, she had suffered from dyspnoea and palpitations, increased by any exertion. About ten days before admission she began to complain of cough, headache, and vomiting, and swelling appeared in her ankles, which gradually extended upwards.

The treatment consisted in the administration of purgatives and diuretics, and particularly the bitartrate of potash and tincture of digitalis, while leeches and mustard and linseed poultices were applied over the right hypochondrium. At first there was a manifest improvement in all the symptoms; but about a fortnight after admission the indications of obstructed cardiac circulation became aggravated: the dyspnoea and dropsy increased, and the lips and face were livid; the jaundice was more marked, the vomiting more urgent, and the motions contained less bile. The pulse was very rapid, and on November 10 was scarcely perceptible. At eleven P.M. of this day the girl died.

On examination of the body, the heart was found much enlarged, weighing 13 ounces. The mitral valve was much thickened and its margins adherent, so that the orifice was contracted, and its circumference measured only fifteen lines. Both lungs were much congested, and presented the ordinary anatomical characters of bronchitis; but they were nowhere consolidated. The peritoneum contained about a pint of clear serum. The liver was very large for the patient's age, weighing nearly 4 pounds. Its outer surface was smooth; and, on section, the branches of the hepatic vein were found gorged with dark blood, contrasting strongly with the intermediate pale-yellow hepatic tissue. On microscopic examination, the quantity of oil in the secreting cells did not seem increased. The spleen weighed $6\frac{1}{4}$ ounces, and was firm and dark on section. The pyramids of the kidneys were much congested, but the renal tissue in other respects was healthy. The mucous membrane of the pyloric half of the stomach presented the ordinary characters of catarrhal inflammation.

As an illustration of congestion of the liver arising

from other causes, I may narrate to you the particulars of the following case :—

CASE XXV.—*Indigestion from Habitual Surfeit—Residence in Tropics—Exposure to Chill—Congestion of Liver.*

Mr. C——, aged 30, a gentleman much addicted to the pleasures of the table, consulted me in June 1867, on his return from India. He had for several years suffered from constipation, flatulence, and a feeling of weight and oppression in the region of the liver. About six weeks before I saw him, he was attacked with pain in the region of the liver, followed by vomiting and jaundice, after sleeping on a verandah in the night air in India. He had leeches applied over the liver, and was ordered home at once. I found him still moderately jaundiced; the liver enlarged, measuring 5 inches in the right mammary line, and slightly tender; no vomiting, but the bowels constipated; a bitter taste in the mouth, and nausea. The motions were light but contained bile. The urine was scanty, dark, contained bile-pigment, deposited much lithates, and became very dark on the addition of nitric acid after heating. He was treated with saline purgatives and occasional pills of the comp. colocynth mass (gr. vi.), podophyllin (gr. $\frac{1}{3}$), and extract of henbane (gr. ii.); an effervescing mixture of citrate of potash was ordered to be taken three times a day; a warm bath three times a week; moderate exercise; and a simple diet, from which alcohol in every form was excluded. At the end of ten days, the patient was much improved, the jaundice had almost gone, and the hepatic dulness diminished. A mixture with nitric acid and compound infusion of gentian was now substituted, and in two or three weeks more the patient had regained his usual health.

In the next case the patient's symptoms were probably those of congestion of the liver, which, had it not been for the treatment adopted, would probably have been accompanied with catarrh of the ducts and jaundice as in her two previous attacks.

CASE XXVI.—*Congestion of the Liver.*

Jemima S——, aged 35, a corpulent married woman of sedentary habits, was admitted into the hospital under my care on November 14, 1866. She had always suffered much from dyspepsia and flatulence, and on two occasions, when she was 21 and 28, she had been laid up for several weeks with vomiting, jaundice, and pain in the region of the liver. On Sunday evening, November 11, probably after some indiscretion in diet, she felt a weight in the stomach. She passed a restless night, and next morning she was seized with pain, rather severe, in the region of the liver, and constant retching; the retching ceased at the end of twenty-four hours, but the pain increased until the time of admission. Her symptoms then were as follows:—Pulse 120. Temp. 99°·2. Slight yellowish tint of conjunctivæ. Hepatic dulness increased, measuring 5½ inches in the right mammary line. Complains of severe pain in the hepatic region, aggravated by pressure or inspiration, or by lying on the left side. Much nausea and flatulence. Urine presents no distinct reaction of bile-pigment, but becomes very dark on the addition of nitric acid after boiling.

Six leeches were applied to the region of the liver, and a draught, containing one drachm of sulphate of magnesia in an ounce of peppermint water, was prescribed to be taken every six hours.

By November 16, the patient had had frequent and copious bilious motions, and the symptoms had begun to subside. A mixture with nitro-muriatic acid and infusion of gentian was now ordered, and on November 30, she left the hospital, with a good appetite, free from pain, and with the hepatic dulness in the right mammary line less than 5 inches.

The next form of painful enlargement of the liver is due to

VI. INFLAMMATION OF THE BILIARY PASSAGES.

This condition is usually associated with more or less congestion of the hepatic tissue, and accordingly its clinical characters are those of congestion, with

those peculiar to catarrh of the bile-ducts and gall-bladder superadded. Thus we have—

1. Enlargement of the liver, which, like that from congestion, is uniform in every direction, and rarely very great; but which is sometimes accompanied by enlargement of the gall-bladder in the form of a more or less pyriform tumour projecting from the anterior margin (see fig. 13, page 145).

2. The portion of liver projecting below the right ribs is smooth on palpation.

3. There is a feeling of tightness and distension in the right hypochondrium, with tenderness on pressure. This tenderness is usually particularly marked over the enlarged gall-bladder.

4. Inasmuch as the bile-ducts are obstructed from the tumefaction of the mucous membrane, as well as from the inflammatory products thrown off from its free surface, the jaundice, after a day or two, is much more intense than in simple congestion, and the motions contain no bile.

5. Here, again, the circumstances under which the attack occurs are of great assistance in diagnosis.

a. In a large majority of cases the attack is preceded by symptoms of catarrh of the stomach and duodenum. The inflammation, in fact, commences in the mucous membrane of the digestive canal, and extends thence to the common bile-duct. Accordingly there are to be noted, in the first place, a furred tongue, loss of appetite, flatulence, nausea or vomiting, pain and tenderness at the epigastrium, and

sometimes diarrhœa, these symptoms being often accompanied by slight pyrexia. After a few days or longer, jaundice appears, and the fever, if present, may subside, although the dyspeptic symptoms remain. Attacks of this sort are very common in children, as the result of eating indigestible food, or of a surfeit ; and, in that case, the jaundice and other symptoms usually subside at the end of ten days or three weeks. They are also not uncommon in persons of more advanced age of a gouty constitution, and more than once I have met with cases of this sort where the frequent vomiting, the emaciation, and the jaundice, persisting for many weeks, have led to the suspicion of cancer, but have soon subsided under the use of purgatives with colchicum and alkalies. Lastly, inasmuch as catarrh of the stomach and duodenum appears sometimes to be induced by a chill, or other atmospheric influence, jaundice from catarrh of the bile-ducts occasionally prevails as an epidemic.

b. Inflammation of the biliary passages may be secondary to congestion or other diseases of the liver, and then its symptoms may be persistent. It is probable that catarrh of the ducts may not only excite congestion of the hepatic tissue, but may result from it. In any case, where congestion of the liver is developed under the circumstances already mentioned, and where, in addition to the symptoms of simple congestion, there is intense jaundice, with an absence of bile from the motions, we may infer

that there is catarrh of the ducts as well as congestion. Other diseases of the liver, also, such as the waxy liver and the hydatid tumour, are occasionally complicated with catarrh of the ducts; and in this way jaundice may appear in the course of diseases of the liver in which it is usually absent. In Case XXX., which is an example of a very rare disease—enlargement of the liver from tubercular deposit—the jaundice was apparently due to inflammation of the common bile-duct.

c. Inflammation of the bile-ducts or gall-bladder may be due to the irritation of gall-stones or of other foreign bodies. Under these circumstances, it will usually be distinguished by a previous history of biliary colic, which, however, was notably absent in the case of one patient who lately died in the wards (Case XXIX.).

d. Certain poisons, such as those of pyæmia and phosphorus, have been recently stated by Virchow to excite catarrh of the bile-ducts.*

Treatment.—The rules already laid down for the treatment of congestion of the liver are also applicable to catarrh of the bile-ducts. Little more need be added, except that—

1. Leeches and cupping are on the whole less necessary. In most cases sinapisms and warm fomentation, with purgatives and alkalies, suffice for subduing the disease. The propriety of employing local depletion must be decided by the degree of pain and amount of congestion existing in each case.

* Virchow's Arch. 1865, xxx. hft. 1.

2. When there is reason to suspect that the affection is of a gouty nature, great benefit will often be derived from the addition of colchicum to the alkalies. In these cases also it will be necessary to adopt such measures as are calculated to correct that disordered condition of the stomach and duodenum, which, if neglected, will before long lead to a recurrence of the hepatic attack.

3. The treatment must occasionally be modified by the presence of other diseases of the liver of which the catarrh of the bile-ducts is merely a complication.

Most of you have had an opportunity of watching the following case of painful enlargement of the liver accompanied with jaundice, apparently due to catarrh of the ducts.

CASE XXVII.—*Painful Enlargement of the Liver, with Jaundice, due to Catarrh of the Bile-ducts.*

Elizabeth L——, aged 21, a maid-servant, was admitted under my care on December 7, 1866. For nine months she had been weakly and unable to take a place, and had also suffered from dyspeptic symptoms. Ten days before admission, at the cessation of the last catamenial period, which had its usual duration, she had been seized with great nausea and vomiting, but she had no diarrhoea. Five days after this she began to complain of pain and tenderness in the region of the liver; but the pain was never very severe. About the same time jaundice made its appearance, which increased in intensity, and was accompanied by much itchiness of the skin.

On admission, there was a deep jaundiced colour of the entire skin and conjunctivæ; the urine was very dark, and gave the characteristic reaction of bile-pigment; the tongue was thickly coated, and there was no appetite, but the vomiting and pain in the side had much subsided; the lower margin of the liver was ascertained to project about an inch below the margin of the ribs

in the right mammary line, and here there was slight tenderness on pressure; the bowels had a tendency to be constipated, and the motions were clay-coloured, without a vestige of bile-pigment; the pulse was 100; the skin rather hot (temperature 100° F.); the respiration was slow and easy; and the physical signs of the heart and lungs were normal.

The treatment consisted in the frequent administration of saline purgatives (sulphate of magnesia), and a blue pill occasionally at bed-time, together with the application of mustard and linseed poultices to the region of the liver.

The bowels were freely purged, and on December 17 the symptoms had considerably improved; the pulse had fallen to 68; the tongue was clean; there was neither nausea nor vomiting; the appetite was returning; and the urine contained less bile-pigment. No change, however, had taken place in the colour of the skin and conjunctivæ, which were still deeply jaundiced. An alkaline mixture, containing bicarbonate of soda, chloric ether, and tincture of orange, was now substituted for the sulphate of magnesia; a purgative was still given occasionally, and the patient had a warm bath twice a week.

On December 20 the jaundiced tint was first noticed to be giving way, and from this date it gradually faded until January 7, 1867, when it had quite disappeared. A tonic mixture with nitric acid and quinine was now ordered, and on January 22 the patient left the hospital in good health.

The following case is cited as a good illustration of catarrh of the bile-ducts occurring in a gouty individual.

CASE XXVIII. — *Gouty Dyspepsia—Jaundice from Catarrh of the Bile-ducts.*

In the autumn of 1865 I was consulted by Mr. C. D——, a gentleman aged 30. His father had been a martyr to gout, and a younger brother had suffered from it early in life. He had never had well-marked gout himself, but he had long been liable to gastric derangements characterized by nausea and flatulence and transient pains in the small joints. About three weeks before I saw him he had been seized suddenly, about an hour after dinner, with a severe pain at the epigastrium, followed by vomiting and nausea.

A few days later jaundice appeared, and gradually increased in intensity; the nausea continued without vomiting, and the patient became much emaciated. On examination I found the lower margin of the liver projecting more than half an inch beyond the edge of the ribs in the right mammary line, and slightly tender on pressure. There was intense jaundice of a deep olive tint, great itchiness of the skin, and complete absence of bile from the motions. The urine was dark like porter. Pulse 60. The patient had no appetite, and had nausea and flatulence after everything he swallowed; he was extremely weak and thin, and his appearance in an older man would certainly have suggested the existence of malignant disease of the stomach or liver.

The treatment adopted consisted in the application of mustard and linseed poultices to the region of the liver, warm baths, blue pill with saline purgatives, a mixture with citrate of potash and vinum colchici and a diet restricted to milk, beef-tea, and farinaceous articles.

After two days the symptoms began to improve, and by the end of three weeks the jaundice had quite disappeared, and the patient was restored to his usual health.

In the following case death was due to uræmia from diseased kidneys, but the hepatic symptoms appeared to result from inflammation of the gall-bladder and bile-ducts excited by gall-stones, which was subsiding before death.

CASE XXIX.—*Inflammation of the Biliary Passages excited by Gall-stones—Gangrene of Foot—Diseased Kidneys—Death by Uræmia.*

Most of you will remember the patient, J. K——, aged 49, who was a patient in Cambridge Ward from October 27, 1866, until his death on November 21. His story was that he had enjoyed good health until the previous June, when he began to suffer from loss of appetite, lowness of spirits, and pain and flatulence after meals. About the same time he got a rusty nail into his left big toe. This resulted in an abscess, which burst, and continued discharging until a few days before admission. He had continued working, however, as a labourer, until within the last

three weeks. During his illness his weight had diminished from 12 st. to 11 st. 5 lbs. On October 20 he had a severe rigor, lasting for three hours, and followed by a rather severe, constant, 'gnawing' pain, with tenderness in the region of the liver, vomiting of bitter green fluid, and headache. Two days later his skin became jaundiced, and he suffered from itchiness of the skin and loss of sleep. About the same time that the jaundice appeared, the left big toe became black, and the ulceration extended. At no time of his life had he suffered from symptoms of biliary colic.

On admission it was noted that the patient had rather deep jaundice of the skin and conjunctivæ. He complained of general itchiness, and of dull pain in the region of the liver, which was uniformly enlarged, the dulness in the right mammary line being $5\frac{1}{2}$ inches. There was also decided tenderness at a spot corresponding to the gall-bladder, which was also enlarged. The abdomen was distended and tympanitic; the ingesta were constantly vomited within half an hour; the tongue was moist, jaundiced, and furred; the bowels were costive, and the motions stone-coloured. The urine was of the colour of porter, and contained a large quantity of bile-pigment and also of albumen, with granular and a few oil-casts. On the dorsum and sole of the left big toe were several large sloughy ulcers, the surrounding soft parts being much swollen and livid. The pulse was 72; the skin was cool; and there had been no rigors or perspirations. The patient was treated with blisters and mustard and linseed poultices to the region of the liver, while bismuth, chloric ether, purgatives, &c., were given internally.

For some time there appeared to be considerable improvement: the jaundice diminished, and bile reappeared in considerable quantity in the motions. But about November 12 the vomiting became more urgent, and the prostration increased. On November 19 the left foot was found to be much swollen, and livid lines marking the course of the lymphatics passed up the legs. On November 20 an abscess was opened above the left ankle, from which fetid pus and gas escaped. On the same day the man was seized with a fit of convulsions, followed by coma. These fits recurred in rapid succession, so that he had nearly thirty before his death at five P.M. on November 21.

On examination of the body after death, the brain and its membranes were found to be normal, except that there was a considerable amount of fluid, which contained urea, at the base and

in the lateral ventricles. The kidneys were considerably enlarged, and there was much fatty and granular deposit in the secreting cells. The liver was large, and weighed 80 ounces; its secreting cells were loaded with oil; the lobules were unusually distinct, giving a granular appearance to the organ on section. The gall-bladder contained a soft, black concretion, as large as a walnut, and many small, irregularly-shaped fragments of the same material. These were suspended in a small quantity of dark-green viscid fluid, which, on microscopic examination, was found to contain a large number of pus-corpuscles. The mucous surface of the gall-bladder had a stretched, white appearance, and at the fundus was deeply injected, granular, and excoriated. The bile-ducts contained a similar viscid fluid to that in the gall-bladder, with minute particles of black inspissated bile. This could be squeezed into the duodenum without much difficulty. The mucous membrane of the stomach and duodenum was minutely injected with numerous small ecchymoses, and the surface was coated with much viscid mucus. There was great œdema and congestion of both lungs. Fat was deposited in large quantity throughout the body, and all the soft tissues were deeply jaundiced.

In the following case also the jaundice appears to have been due to inflammation of the biliary passages, which was subsiding before the patient's death. But the main interest of the case lay in the cause of enlargement; for although Rokitansky* speaks of hepatic tubercle occurring 'in the shape of semi-transparent, greyish, crude, miliary granulations, in which case it is more especially the product of acute tuberculosis,' tubercle has not usually been regarded as one of the causes of hepatic enlargement. Frerichs also mentions the occurrence of nodules of yellow tubercle in the liver, which may soften into vomicæ; while other observers have noted contractions and

* Path. Anat. Syd. Soc. Transl. vol. ii. p. 149.

dilatations of the fine bile-ducts from the deposit of tubercle in their walls.*

CASE XXX.—*General Tuberculosis—Enlargement of the Liver from Tubercular Deposit—Jaundice from Catarrh of the Bile-ducts—Embolism of the Spleen.*

Mary C——, aged 40, was admitted into the Middlesex Hospital, under my care, on December 17, 1867. Her father and mother had both died at the age of 50, of some chest affection, and of eleven brothers and sisters all were dead but one, though the patient could not say of what they had died. The patient was extremely prostrate, and somewhat confused in her mind. So far as her history could be obtained, it was to the effect, that six months before she had lost her appetite, and vomited about half an hour after every meal. Two or three months after this she became jaundiced. She had not suffered from cough, hæmoptysis, rigors, or night-sweats, but from the first she had lost flesh and strength.

On admission there was jaundice of moderate intensity of the skin and conjunctivæ, and the urine exhibited the reaction of bile-pigment, and threw down a copious deposit of lithates, but contained no albumen. There was no itchiness of the skin; tongue dry and brown, except at the edges, which were preternaturally red. The patient stated that up to the time of admission she had vomited almost everything within half an hour of swallowing it, but she did not vomit once after admission. A motion passed soon after admission was formed, and of a dark brown bilious colour. The hepatic dulness was increased, in the right mammary line measuring 5 inches, and extending fully an inch below the margin of the ribs. The portion below the ribs felt smooth and was slightly tender. Pulse 120, small and feeble; a faint systolic bellows' murmur at left apex of heart; temperature 100°·2. There was nothing to attract attention to the lungs, which in the patient's weak state were not examined. A large superficial bed-sore over sacrum.

The patient was treated with bismuth, chloric ether, and stimulants, but she became rapidly more prostrate; low muttering de-

* Frerichs' Dis. of Liv. Syd. Soc. Ed. ii. 220.

lirium set in, the motions and urine were passed in bed, and death took place on December 23.

Autopsy.—About a pint of clear serum was found in the peritoneum. The liver was very large and weighed 77 oz. Its capsule was not thickened or adherent. Its surface was generally smooth, but marked by numerous minute depressions and elevations. The glandular tissue was pale yellow and opaque, exactly like that of a fatty liver, from which it differed, however, in being remarkably firm and tough. On section, a little thin watery bile could be squeezed from the divided bile-ducts, many of which presented small dilatations. The gall-bladder contained a small quantity of a similar fluid, as well as numerous minute, black, gritty concretions. On microscopic examination it was ascertained by Dr. Cayley that the enlargement of the liver was mainly due to the presence of numerous miliary tubercles scattered through the glandular tissue between the lobules, and presenting all the structural characters of grey tubercle.

The mucous membrane of the stomach was pale and normal, but immediately below the pylorus that of the duodenum, for about 8 inches, was intensely injected, tumid, and studded with numerous small granular punctated elevations, apparently enlarged solitary glands. The lining membrane of the common bile-duct was also very red, and the mucous membrane slightly swollen, but the passage was not obstructed. There were three small tubercular ulcers in the lower part of the ileum. Both lungs were studded with numerous grey miliary tubercles, and near both apices was a small patch of old grey tubercle. The edge of one of the flaps of the mitral valve was much thickened. There was no lymph at the base of the brain, and no tubercles in the pia-mater, but there was much serous fluid beneath the arachnoid and in the cerebral ventricles, and in the cavity of the arachnoid over both hemispheres was a thin film of extravasated blood. In the uterus there was a fibrous tumour as large as a cocoa-nut, and the position of the right ovary was occupied by a tumour as large as an orange, partly solid, and partly breaking down into a soft cheesy material. The right Fallopian tube was as large as a finger, and filled with soft putty-like material; its lining membrane was rough and ulcerated, like that of the pelvis of the kidney in tubercular pyelitis. The spleen was large, and weighed $15\frac{1}{2}$ oz.; it was very soft, and studded with numerous abscesses, from a pea to a hazelnut in size, and containing thick yellow pus; there were also

several solid deposits in the spleen, having the characters of recent embolism. The cortices of both kidneys were studded with minute yellow tubercular granules.

The next form of enlargement of the liver, attended by pain and jaundice, to which I wish to direct your attention, is—

VII. ENLARGEMENT FROM OBSTRUCTION OF THE COMMON BILE-DUCT BY CALCULI, TUMOURS, ETC.

Obstruction of the common bile-duct may lead to enlargement of the liver in two ways.

a. By causing dilatation of the biliary passages with accumulation of bile in them. But if the obstruction be of long standing, the liver may ultimately contract to less than its natural size, its secreting tissue becoming atrophied from the pressure of the permanently distended bile-ducts.

b. By inducing inflammation of the biliary passages, associated with more or less congestion of the hepatic tissue.

The distinguishing characters of the enlargement of the liver that occurs under such circumstances are as follows :—

1. The enlargement is rarely great, and, with one important exception, it is uniform in every direction. The exception referred to is due to the enlargement of the gall-bladder, which can often be felt as a pyriform tumour projecting from the lower margin of the liver. This enlargement is due, in the first place, to an accumulation of bile, but after a time not unfrequently to the admixture or substitution of

inflammatory products. The late Dr. Bright has recorded a case in which such an enlargement of the gall-bladder formed an oval tumour descending nearly to the crest of the ilium; and you have had an opportunity of examining a similar though smaller tumour in the case of J. W——. (Case XXXI. and fig. 13.)

2. There is jaundice, which, if the cause of obstruction be a gall-stone, like the pain about to be referred to, is often in the first instance paroxysmal, but by the time that the liver becomes enlarged is permanent and usually intense, and is accompanied by a total disappearance of bile-pigment from the motions. In cases of persistent jaundice, where from the colour of the motions it is clear that the flow of bile into the bowel has been cut off for many weeks, there can be little doubt that there is obstruction of the common duct; and if the jaundice has been at first paroxysmal, the cause of that obstruction is probably an impacted gall-stone. But if there be no evidence of the jaundice having been paroxysmal, it may be difficult to say whether the obstruction be due to an organic obliteration of the duct at its duodenal opening from an ulcer or from a cancerous growth in the duodenum, or to a tumour in some other part of the course of the duct, or to pressure by a tumour on the duct from without. The rules for your guidance under these circumstances will be best considered when I come to describe the various forms of jaundice arising from obstruction of the common bile-duct.

3. Pain and tenderness in the region of the liver, and particularly in the situation of the enlarged gall-bladder, are present in most cases. The pain is always greatest in those cases where there is catarrh of the biliary passages, or where the bile-duct is compressed by a tumour which at the same time compresses and stretches the hepatic plexus of nerves. When the obstruction is due to the impaction in the duct of a gall-stone, there will usually be a history of attacks of paroxysmal pain with the other phenomena of biliary colic.

4. The diagnosis will usually be assisted by the presence of those symptoms which mark the various morbid conditions producing obstruction of the bile-duct, and which will be considered hereafter under the head of Jaundice.

The *treatment* of this form of enlargement of the liver, or rather of its various causes, will also be best considered under the head of Jaundice.

In the meantime, I may recall to your recollection the following case, which has been under your observation for some weeks, and which is a good illustration of enlargement of the liver and jaundice, apparently from gall-stones, except that the patient's age is considerably under that at which gall-stones are ordinarily met with. In this case also, the enlargement of the gall-bladder and many of the other symptoms appeared to be due to catarrhal inflammation of the bile-ducts and gall-bladder, excited by a gall-stone.

CASE XXXI.—*Enlargement of Liver and Dilatation of Gall-bladder from Obstruction of Common Duct by a Calculus.*

John W——, aged 30, a stone-cutter, was admitted on February 5, 1867. He had enjoyed good health until six months before he came to hospital, when he began to suffer from acute paroxysms of pain in the abdomen. For a week he would have several paroxysms daily; then he would be free for a week, and during this in-

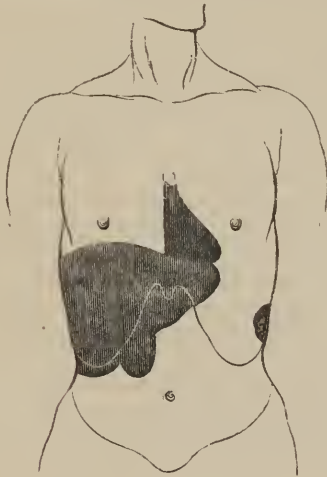


Fig. 13 shows the enlargement of the Liver and the Tumour in the case of J. W., on February 20. Compare this with Fig. 3, at p. 5.

terval he would be able to resume his work. The attacks were not accompanied by vomiting, but the first was followed by jaundice, which has never left him. The paroxysms of pain continued to recur for six weeks, but subsequently to this he had none; he had suffered much, however, from flatulence and itchiness of the skin, and had lost flesh. On admission, there was universal jaundice of moderate intensity: the urine was loaded with bile-pigment, but the motions contained none. The hepatic dulness was mode-

rately and uniformly increased, measuring five inches in the right mammary line. No tumour corresponding to the gall-bladder could be discovered, but possibly this was obscured by the flatulent distension of the bowels. There was no ascites. The tongue was moist, and but slightly furred. The patient's appetite was good, and he never vomited; but he was obliged to be very careful as to his diet, as he suffered much from flatulence and pain after eating. The pulse was 72.

About a fortnight after the patient's admission he became much worse; and on February 20 it was noted that the jaundice was more intense, that the urine was darker, and that the hepatic dulness was increased, measuring fully $5\frac{1}{2}$ inches in the right mammary line. In addition, there was now in the situation of the gall-bladder a distinct tumour (see fig. 13), extending $1\frac{1}{2}$ inch below the margin of the liver, and measuring $2\frac{1}{2}$ inches transversely. This was tender on pressure. The temperature had risen to $104^{\circ}2$ F., and the pulse to 96. The tongue was somewhat dry, and the motions were perfectly devoid of bile. These symptoms continued, with occasional vomiting, for several days; but on February 25 the temperature had fallen to $99^{\circ}2$, and on the 27th to 97° . On March 1 the pulse was down to 72, and the tumour in the region of the gall-bladder had disappeared. On March 4 the motions contained abundance of bile, and the jaundice was fading. By the beginning of April the jaundice had almost disappeared; and in May the patient was able to resume his employment.

The motions were carefully searched for gall-stones for ten days subsequent to February 24, but none were found. Possibly a gall-stone may have either become disintegrated, or slipped back into the gall-bladder. During the acute stage the patient was treated with alkalies, ammonia, ether, belladonna, and opium. During convalescence, strychnia appeared to relieve the flatulence, and the disappearance of the jaundice was encouraged by warm baths and diaphoretics.

LECTURE V.

ENLARGEMENTS OF THE LIVER.

PYÆMIC ABSCESES—TROPICAL ABSCESS.

GENTLEMEN,—The first form of enlargement of the liver to which I desire to draw your attention to-day is that due to

VIII. PYÆMIC ABSCESES.

The abscesses which are often developed in the liver in the course of pyæmia are for the most part many in number and small, and in these respects they differ from the tropical abscess, which is usually single, and often attains a large size, so as to form a distinct tumour. The clinical characters vary in accordance with this anatomical difference, and with the different conditions under which the hepatic disease occurs. Those of the pyæmic abscess are as follows :—

1. There is enlargement of the liver; usually of moderate extent, but sometimes so great that the lower margin of the organ reaches to the umbilicus.

2. The enlargement is uniform in every direction, and does not produce any bulging of the ribs. In exceptional cases only, one of the abscesses enlarges

somewhat more than the others and forms a small bulging tumour at the epigastrium; and in cases of still rarer occurrence, the lower margin of the liver, as felt through the abdominal parietes, has a noded character, from the presence of several small abscesses or inflammatory deposits along its free margin.

3. No fluctuation can be felt in the enlarged liver. The abscesses are rarely large enough to admit of this. Only in those rare cases where one of the abscesses enlarges so as to form a bulging in the epigastrium, or where a small quantity of pus becomes encysted between the liver and abdominal wall (Case XXXIV.), is anything approaching to fluctuation perceptible.

4. Pain and tenderness are always present. They are often among the first symptoms noted, and are usually acute in consequence of inflammatory action being propagated from certain of the abscesses to the superimposed peritoneum. The pain is often increased by coughing or by a long inspiration, so that in consequence the respirations are quick and short, and mainly thoracic.

5. Jaundice is present in the majority of cases—in fully four-fifths; but the possibility of its absence must be kept in view in diagnosis. The intensity of the jaundice varies. In most cases it appears to be due to the morbid condition of the blood to which the term pyæmia is applied, just as jaundice is known to result from many other blood-poisons, and then

it is usually slight, and the motions are still tinged with bile-pigment ; but if the pyæmia arise from an ulcer of the biliary passages, excited by the pressure of an impacted gall-stone, the jaundice may be intense and the excrement devoid of bile-pigment.

6. Pyæmic abscesses of the liver rarely interfere with the portal circulation. Accordingly there is no enlargement of the veins of the abdominal parietes, and only in exceptional cases (from implication of a large branch of the vein), ascites. The spleen, it is true, is usually enlarged, but this is due not so much to obstructed circulation as to the tendency of the spleen to enlarge in consequence of the morbid condition of the blood, as happens in the course of many diseases arising from a blood-poison. Occasionally fluid is thrown out into the peritoneum as the result of peritonitis.

7. The constitutional symptoms are important in diagnosis. They are mainly those of hectic fever, with increased temperature, emaciation, progressive prostration, and a tendency to vomiting, diarrhœa, and the 'typhoid state.' Rigors and profuse perspirations during sleep are common, and afford material assistance in diagnosis ; but it is well to remember that they are not necessary symptoms. The rigors occasionally recur at such regular intervals that the attack simulates ague ; errors in diagnosis are constantly committed from this fact not being remembered (Case XXXIII.). On the other hand, the possibility of rigors resulting from the mere passage

of a gall-stone must not be lost sight of. As the disease advances, symptoms of blood-poisoning, such as a dry brown tongue and delirium, make their appearance.

8. The course of the disease is rapid, usually ranging from two or three weeks to three months. I have never known the latter limit exceeded. This rapid course may be of service in diagnosing cancer, in which the duration is more protracted, from pyæmic abscess of the liver.

9. The diagnosis will also be assisted by keeping in view the circumstances under which the disease usually occurs. Among them the following hold a prominent place :—

a. External injuries and surgical operations. When symptoms like those above described follow either of the causes now mentioned, there need be no difficulty about the diagnosis. The most of the cases, however, which come under the care of the physician depend upon internal causes, and then the difficulty of diagnosing is increased.

b. Ulceration of the stomach or intestine. I have repeatedly known pyæmic abscess of the liver supervene on simple ulcer of the stomach, and shall relate to you immediately the particulars of a case where this occurred. The same condition of liver may follow simple ulceration of any portion of the intestine, or even cancerous ulceration of the stomach or bowel. Pyæmic deposits in the liver, however, only occur in exceptional cases of intestinal ulceration,

probably for the same reason that general pyæmia only occurs in exceptional cases of external injury (see pp. 162-3).

c. Ulceration of the gall-bladder or of the bile-ducts may give rise to pyæmic abscesses of the liver, which in this way may be a sequel of gall-stones. I shall narrate to you presently a case where what appeared to be an ordinary attack of biliary colic was followed by fatal inflammation of the liver (Case XXXV.). When the common bile-duct also is obstructed by a gall-stone or from any other cause, the ducts in the interior of the liver may become dilated into irregular cavities full of pus, and give rise to many of the symptoms of pyæmia.

d. In a former lecture I brought before your notice two instances in which a suppurating hydatid cyst appeared to be the starting-point of pyæmic abscesses in the liver (see pp. 99 and 100).

e. Lastly, any suppurating ulcer or cavity on or near the outer surface or in the interior of the body, especially if in connection with diseased bone or communicating with the external atmosphere, may induce pyæmia with secondary deposits in the liver. On more than one occasion, for instance, I have found these deposits in the liver resulting from a tubercular vomica in the lungs, ulcerative endocarditis, pyelitis, &c.

When the signs and symptoms already enumerated supervene on those of any of the maladies now referred to, the probability of pyæmic abscess of the liver ought at once to suggest itself. But occasionally

the primary disease is latent, and the first symptoms are those of inflammation of the liver. Even then, however, the probability of pyæmic abscess ought to suggest itself in English practice, inasmuch as, with extremely rare exceptions, this is the only form of hepatic abscess met with in this country in persons who have never been abroad.

Treatment.—In pyæmic abscess of the liver, medical art, it is to be feared, is powerless to avert the fatal result, and can only mitigate the patient's suffering.

1. By hygienic arrangements, by the local use of carbolic acid in open sores and wounds, and by evacuating decomposing pus pent up in any part of the body, much can be done in the way of preventing general pyæmia in surgical injuries; but, unfortunately, in a large number of cases of pyæmic abscess in the liver that come under the physician, the primary disease is inaccessible.

2. Depletion, both general and local, is contra-indicated; but if the pain be very acute it will often be materially relieved by the application of a few leeches to the region of the liver. Mustard and linseed poultices are also useful for relieving the pain.

3. Professor Polli, of Milan, has of late years written strongly in favour of the sulphites of potash and soda as antidotes for the pyæmic poison. The power which these substances possess of arresting putrefaction or fermentation out of the body it is believed that they can exercise in the living blood. I have tried them repeatedly, and I regret to say

that in my practice they have signally failed. They may be given in doses of twenty or thirty grains every four hours.

4. Quinine and mineral acids have appeared to me to be the remedies most generally useful. They support the patient's strength, keep the tongue moist, and tend to diminish the profuse sweating.

5. Opium or morphia in repeated doses will be necessary in most cases to relieve pain or procure sleep. If there be much retching, the subcutaneous injection of morphia will be preferable to administering opiates by the mouth.

6. The treatment must often be modified in such a way as to counteract various distressing symptoms which are apt to arise, and more especially vomiting and diarrhœa. For the vomiting, the best remedies are ice, bismuth, effervescing alkaline draughts, and the application to the epigastrium of sinapisims or of a small blister, followed by the sprinkling of a quarter of a grain of morphia on the blistered surface. For the diarrhœa you must have recourse to vegetable and mineral astringents, and particularly the acetate of lead and morphia, and opiate enemata and suppositories.

7. The diet must be of as nutritious a character as is compatible with the patient's digestive powers. It ought to consist of such articles as milk, beef-tea, and eggs, given frequently, but in small quantities at a time. In most cases it will be necessary to give small quantities of wine or brandy, which ought to be well diluted.

I shall now proceed to relate to you the particulars of a few cases in illustration of the foregoing remarks. In the first case the hepatic disease was the result of an external injury.

CASE XXXII.—*Injury of Cranium, followed by Pyæmia and Multiple Abscesses in the Liver.*

Thomas D——, aged 21, was admitted into one of the surgical wards on August 16, 1867, with lacerated wounds of the scalp, a fracture of the sixth left rib, and a bruise of the left shoulder—injuries which he had received from being run over by a cab. He had so far recovered that on September 3 he was able to be out in the garden; but on the same day he was seized with rigors, followed by febrile symptoms, headache, and loss of appetite. During the next two days he had several attacks of severe rigors, like those of ague, followed by moderate perspiration and frequent vomiting.

When he first came under my care, on September 6, he had all the symptoms of blood-poisoning, but without any eruption on the skin. Pulse 120; respirations 36; temperature 103°. Alternate fits of chilliness and perspiration. Countenance heavy and depressed; great lassitude; throbbing headache, but mind quite clear; great prostration, and tendency to syncope on sitting up; frequent retching, with tenderness in the epigastrium and right hypochondrium. The tongue was moist, and but slightly furred. The bowels had been freely opened by medicine. The cardiac and respiratory signs were normal. The urine contained a small quantity of albumen, with blood-corpuscles and epithelial casts of the uriniferous tubes. A wound in the left temporal region of the scalp was found covered with a hard scab, from beneath which about a teaspoonful of dirty, not fetid, pus could be squeezed. Soon after the patient's admission he became very restless and delirious; there was no paralysis, but the hearing was preternaturally acute. The tongue became dry and brown, and there was frequent vomiting with a tendency to diarrhoea. The tenderness in the epigastrium and right hypochondrium continued, and the hepatic dulness became much increased, extending down almost to the umbilicus. The surface of the organ felt smooth. The skin was sallow, but there was no decided jaundice.

The patient was treated mainly according to the plan recom-

mended for pyæmia by Professor Polli, of Milan, with large doses of sulphites. Sulphite of soda was given in doses of fifteen grains every four hours. No improvement, however, was observed; and the symptoms above noted continued almost till death, at 9.45 P.M. on September 9.

On examination of the body after death about a square inch of bone, corresponding to the wound in the scalp, was bare and discoloured. The bone appeared scratched on the surface. It was not fractured; but between its under surface and the corresponding dura-mater there was about a drachm of pus. The veins leading from this to the longitudinal sinus contained pale, soft, non-adherent coagula. The liver was very large, extending down to the umbilicus, and weighing 104 ounces. Its tissue was dark and intensely injected, and riddled with innumerable pyæmic deposits breaking down into pus, from the size of a pin's head up to that of a walnut. The spleen was large, weighed $10\frac{1}{2}$ ounces, and was dark and firm, but contained no infarctions. Both kidneys were much enlarged, weighing together $18\frac{1}{4}$ ounces. Their surfaces were smooth, and the capsules non-adherent. The cortical substance was greatly hypertrophied and deeply injected, but was free from pyæmic deposits. The sixth left rib was fractured at about two inches from the cartilage. The edges overlapped, and were enveloped in callus; but there was no trace of laceration of the lung, or of pleurisy—old or recent—in the neighbourhood. There were slight traces of recent pericarditis, and numerous minute ecchymoses beneath the pericardium.

In the second case the hepatic inflammation followed a simple ulcer of the stomach.

CASE XXXIII.—*Multiple Abscesses in the Liver Secondary to Simple Ulcer of Stomach.**

John P—, aged 51, was admitted into the London Fever Hospital on October 6, 1865. For six weeks he had been suffering from pain, tenderness, and flatulence in the abdomen after food, followed occasionally by vomiting. He had suffered from similar symptoms on former occasions, but had always recovered. The

* A second case of a similar nature is recorded by me in the Path. Trans. vol. xvii. p. 146.

hepatic dulness was $4\frac{1}{2}$ inches in the right mammary line. There was no jaundice. Pulse 84. Bismuth and a milk diet were prescribed. Three days after admission it was noticed that the patient had a daily febrile accession about one P.M.; and it was ascertained that twenty-two years before (but never since then) he had suffered from ague in Kent. Quinine was accordingly administered in large doses. It had no effect, however, on the paroxysms. On the contrary, they became more severe, came on at irregular intervals, and were followed by profuse perspirations and great prostration. The tongue also became dry and brown, the pain and tenderness at the epigastrium were greatly increased, and the bowels became very loose. On October 16 it was noted that he was much lower and greatly emaciated, and that the skin and conjunctivæ had a decidedly jaundiced tint, although the motions contained plenty of bile. The hepatic dulness in the right mammary line was now $5\frac{1}{2}$ inches; but the enlargement was uniform, and free from nodulation. There was considerable tenderness on pressure below the lower margin of the right ribs. The splenic dulness was increased. Pulse 96; temperature 101° . The symptoms above narrated became gradually aggravated. He still had irregular paroxysms of rigors, followed by fever and sweating. On October 21 the jaundice was noted as deep, although bile was still present in the motions. The mind was slightly confused, and he had occasional low delirium. He gradually sank, and died on October 24.

On post-mortem examination, near the pyloric end of the stomach, on its lower and posterior surface, was a circular ulcer the size of a crown-piece, with its edges slightly elevated and indurated, but containing none of the microscopic elements of cancer. From the base of this ulcer a small fistulous channel passed into an abscess, almost the size of a walnut, in the head of the pancreas. The liver generally was enlarged, and weighed 81 ounces; the posterior half of the right lobe was studded with minute abscesses, from the size of a pin's head up to that of a pea, containing thick yellow pus. The intervening hepatic tissue was very hyperæmic. There was no peritoneal inflammation over the surface of the liver. The spleen was large, dark, and firm. The other organs were healthy.

In the next case a cancerous ulcer of the stomach appeared to be the exciting cause of the disease in

the liver. The case has additional interest from the fact that there was a small fluctuating tumour at the epigastrium caused by a circumscribed collection of pus between the liver and abdominal parietes.

CASE XXXIV.—*Cancerous Ulcers of the Stomach followed by Pyæmic Abscesses of the Liver.*

In June 1867 I was requested by Dr. Rogers, of Dean Street, to see a patient under his care. He was a man, aged 45, whose father and sister were said to have died of cancer. For several months he had been losing flesh, and had suffered pain after food, and other symptoms of indigestion, but not vomiting. About May 19 his symptoms became worse, and he first consulted Dr. Rogers. He then began to suffer from a constant pain in the right side, febrile symptoms, dyspnoea, and a frequent dry cough, and on May 23 and again on the 28th he had severe attacks of vomiting. About June 2 a slight swelling was first noticed in the epigastrium, and he became slightly jaundiced, and when I saw him on June 8 with Drs. Anstie and Rogers, there was considerable jaundice, with great emaciation and prostration. The pulse was quick and feeble, and there was a tendency to nocturnal perspiration, but no rigors. The tongue was moist, clean, and red; there was no vomiting or diarrhoea, and the motions contained bile. The liver was much enlarged, and in the epigastrium there was a very painful prominent tumour, about the size of half an orange, extremely elastic, and indeed apparently fluctuating. An exploratory puncture was made into this tumour, but only a few drops of blood came away. The patient became daily more emaciated and prostrate; the tongue became dry and brown, and the jaundice increased; although the stools still contained bile-pigment. On June 24 he died from exhaustion. Throughout there had been no rigors, and only slight perspiration during sleep.

On examining the body the liver was found of almost twice the normal size; there were signs of recent peritonitis over its outer surface; the glandular tissue was extremely congested, and was studded with inflammatory (not cancerous) deposits up to the size of a walnut, which were pale yellow, granular, and very friable, but which had not yet softened into pus. Between the left lobe and the abdominal wall there was about an ounce of pus circum-

scribed by firm adhesions. This accounted for the fluctuating tumour felt during life; the fine trocar had probably passed through the abscess into the liver, and thus no pus had been obtained by the puncture. On opening the stomach an ulcer was found about 2 inches from the pylorus; the edges and base of this ulcer were indurated from what the microscope showed to be cancerous tissue, and the surface of the ulcer was ragged and sloughy.

The next case which I shall refer to is that of a lady, 23 years of age, whom I saw in consultation with the late Mr. Young, of Sackville Street, in November and December 1861. It affords an illustration of pyæmic abscesses of the liver supervening on gall-stones.

CASE XXXV.—*Attacks of Biliary Colic followed by Pyæmic Abscesses in the Liver.*

On November 30, 1861, I was called to see Mrs. —, aged 23, who had been married only four or five months. Two years before, she had suffered for several weeks from jaundice, with severe attacks of biliary colic. Ten days before I saw her the jaundice had returned, and during the same period she had been suffering from severe paroxysms of pain in the right hypochondrium, often accompanied by vomiting. Although, notwithstanding the patient's age, her history was clearly one of gall-stones, yet, after making allowance for her hysterical temperament, the symptoms led to the suspicion that there was something more. The pulse was 100, and there was an unusual amount of tenderness in the region of the liver, and particularly in the situation of the gall-bladder. The hepatic dulness was increased; there was also great increase of the splenic dulness. The jaundice was of moderate intensity; and the motions, though very pale, were not entirely devoid of bile-pigment. Leeches, followed by warm fomentations, were applied to the right hypochondrium, and repeated doses of opium were prescribed.

During the first week in December the patient had frequent attacks of vomiting, and on the 4th she miscarried, at the third month. After this she became much worse. She had repeated attacks of rigors, lasting for half an hour or more, and often

followed by the involuntary discharge of light-yellow fluid from the bowels. She had also frequent and severe paroxysms of retching, and the pain in the right side became so intense that she could not take a long inspiration without crying out. The patient was never free from pain and tenderness in the region of the duodenum, but the intense pain was decidedly paroxysmal; sometimes, but not always, the paroxysms seemed to be induced by the patient moving or taking a long inspiration. The pulse varied from 100 to 120; the cheeks were flushed, but there were no perspirations; the patient suffered much from thirst, but even fluids were at once rejected from the stomach. The jaundice diminished; the motions always contained bile, and at last were almost natural in appearance. All treatment failed to give relief; she became rapidly emaciated, and was occasionally delirious during the night; and towards the end the tongue was dry and brown, and sordes collected on the lips and teeth. Death took place on December 23.

On post-mortem examination the liver was found to be large, and the entire substance of both lobes studded with an immense number of circumscribed abscesses, varying in size from a pea to a small orange, and filled with yellow flaky pus. The outer surface was glued by recent lymph to the diaphragm and adjoining organs. The hepatic and common ducts were pervious, and contained bile. The gall-bladder was collapsed, its cavity being scarcely larger than a hazel-nut, and its coats much thickened. A gall-stone, somewhat larger than a pea, was found impacted at the commencement of the cystic duct, and the mucous membrane in contact with the concretion was ulcerated, and partly converted into a blackish slough. Beyond this the cystic duct was obliterated. The gall-bladder contained about a dozen calculi of smaller size, but no bile. The fundus of the bladder was firmly adherent to the duodenum, and between these two viscera was a closed cavity containing gall-stones equalling in size and number those found in the gall-bladder itself. The corresponding mucous surfaces of the duodenum and of the gall-bladder were marked by an extensive cicatrix. These appearances were probably the result of a direct passage of gall-stones through the fundus of the gall-bladder into the bowel in the attack two years before death. The mucous membrane of the first three inches of the duodenum was intensely injected, but not ulcerated. The inner surface of the stomach and intestines presented nothing abnormal. The spleen was four times its normal size. In addition to the coating

of recent lymph, the capsule of the liver at several places presented old thickening and firm adhesions. The right lung and pleura were normal.

Case XXXVI. was remarkable for the large size attained by the liver, and for the absence of any cause of the hepatic inflammation, excepting the softening tubercle in the mediastinal glands.

CASE XXXVI.—*Multiple Abscesses of the Liver—Softening Tubercle in Mediastinal Glands.*

Ann C——, aged 57, a cook, was admitted into the Middlesex Hospital under my care on January 13, 1868. Her father and mother had both lived to upwards of 80, and, with the exception of an umbilical hernia and a great tendency to vertigo, she herself had always enjoyed good health until her present illness, which commenced a week before Christmas with acute pain in the region of the liver, stretching round the back to the left side. This pain was accompanied by febrile symptoms, loss of appetite and sleep, and by a swelling and tightness in the upper part of the abdomen, which increased daily. On January 5, her face and eyes had been noticed to be slightly yellow.

On admission the patient was an extremely corpulent woman, whose skin and conjunctivæ presented a slightly jaundiced tint, and who was so weak as to move her great bulk with difficulty in bed. The abdomen was enormously enlarged, measuring 53 inches at the umbilicus. There was moderate œdema of both lower extremities; but no distinct thrill of fluid could be detected in the abdomen, and percussion yielded a clear sound in both flanks. The great size of the abdomen appeared due partly to an enormous subcutaneous deposit of fat, and partly to enlargement of the liver, which measured 9 inches in the right mammary line, and which reached fully 5 inches below the margin of the right ribs. So far as an examination could be made through the thickened abdominal parietes, the enlargement of the organ appeared to be uniform in every direction, and its surface was hard and smooth. On pressure over it there was decided tenderness, and a pain shooting from the point of pressure to the back. The tongue was dry and red down the centre; much thirst; no vomiting, and bowels regular. Pulse

108. Heart's sound very feeble, but no bellows-murmur. Respirations embarrassed and thoracic; sonorous râles at bases of both lungs. Urine of a dark amber colour, with a copious deposit of lithates, but containing no albumen. Mind clear. Temperature 98° Fahr.

The patient was ordered the day after admission a draught containing a drachm of sulphate of magnesia three times a day, but on January 15, after three doses, the bowels were so purged that a mixture of nitro-hydrochloric acid and gentian was substituted. The diarrhoea, however, persisted, the motions being watery and dark-brown; the tongue continued dry, the temperature rose to 101°·4; on the nights of January 17 and 18, the patient had much low delirium; and in the afternoon of January 19, she died suddenly by syncope while attempting to get out of bed.

Autopsy.—The layer of fat at some parts of the abdominal parietes measured fully 4 inches in thickness. The peritoneum contained about three pints of turbid serum, with small flakes of lymph. The liver was enormously enlarged, its lower margin projecting about 5 inches beyond that of the right ribs. It weighed 256 ounces, and was studded throughout with innumerable minute abscesses, the projection of which from the outer surface gave to this a coarsely granular aspect. The portions of hepatic tissue which remained were in an advanced stage of fatty degeneration, but there was scarcely a quarter of an inch of the organ free from purulent deposit. The gall-bladder was much distended with innumerable black polygonal concretions, from the size of a small cherry to that of a grain of sand. The majority were small, and resembled the grains of coarse gunpowder. The larger ones were found on section to be white internally, and to be composed of cholesterine. The common bile-duct was patent, and after careful examination, no ulceration could be discovered in the lining membrane of the gall-bladder or of any of the ducts, nor in the mucous membrane of the stomach or intestines. There was no pus in the portal vein or embolism of the hepatic artery. The spleen was large and soft. The kidneys were rather large and pale, but appeared normal. The right Fallopian tube was dilated into a cyst the size of an orange, containing a dark thin fluid, and with several small vegetations attached to its lining membrane. There was a fibrous tumour the size of a walnut in the walls of the uterus. At the apices of both lungs there were old tubercular cicatrices, but no

cavities, and in the anterior mediastinum were two or three collections of pus, formed by suppuration of tubercular lymphatic glands. The heart was pale, flabby, and friable, and in an advanced stage of fatty degeneration.

IX. TROPICAL HEPATITIS AND ABSCESS OF THE LIVER.

The pathology of tropical abscess of the liver has been a subject of much discussion, and one on which opinions are still divided. The frequent coexistence in the tropics of abscess of the liver with dysentery has naturally led pathologists to connect the two lesions, some, like Annesley, maintaining that the dysentery is the result of the hepatitis, others that the hepatitis is the result of the dysentery, while a third class, like Dr. Abercrombie, have suggested that the frequent concurrence of the two maladies is merely the result of accident. The doctrine which is now most generally accepted is that propounded more than twenty years ago by Dr. G. Budd, viz. that the hepatic inflammation is the result of purulent absorption from the ulcerated colon, or in fact that the pathology of tropical abscess is identical with that of the pyæmic abscess of this country.*

Considering how frequently in this country abscess of the liver is secondary to ulcers of the stomach or bowels, or other sources of purulent absorption, it would indeed be extraordinary if dysenteric ulceration of the colon never led to a like result, as some have contended. The fact that fatal dysentery with ulce-

* Dig. of Liver, 3rd ed. p. 82.

ration uncombined with hepatic abscess is a common occurrence in India is no argument against hepatic abscess occasionally resulting from dysentery, any more than that, in Europe, pyæmic abscess only occurs in exceptional cases of intestinal ulceration, or of the other sources of purulent absorption already enumerated. Something more than an open sore is necessary for the formation of pyæmic deposits. The discharges from the sore must be in a peculiar state of decomposition. The causes of this decomposition may vary, but where there is no such decomposition there is no pyæmia.

But a large number of the abscesses of the liver met with in tropical countries cannot be ascribed to dysentery, or to a pyæmic origin, or to mechanical injury. Fourteen years ago, I stated that this was the result of my observations on the diseases of Burmah,* and the facts, which have since been published by Morehead,† Bristowe,‡ Frerichs,§ and others, appear to me to be perfectly conclusive on the matter. These facts are of a threefold nature.

1. Cases are not uncommon in tropical countries where there has been abscess of the liver, and where the patient has recovered without any symptoms of dysentery before, during, or after the hepatic malady.

* *Observ. on the Climate and Diseases of Burmah.* Edin. Med. and Surg. Journ. 1854, pp. 245-7.

† *Researches on Diseases in India*, 1856, ii. p. 10.

‡ *Path. Trans.* 1858, ix. p. 250.

§ *Frerichs, Treatise on Dis. of Liver*, Eng. Ed. ii. p. 116.

I shall give you the particulars of such a case immediately (Case XL.).

2. In not a few cases where there has been a concurrence of hepatic abscess and dysentery, the symptoms of the former malady have preceded those of the latter. A case of this sort was recorded by me in the eighth volume of the 'Pathological Transactions' (p. 237), and similar cases are referred to by Morehead, Waring, and Bristowe.

It may perchance be argued that in the cases included under these two heads dysenteric ulceration was really present, but that its symptoms were latent. Dr. Dickinson, for instance, has recorded a case where extensive dysenteric ulceration and a large abscess of the liver were found after death without any symptoms during life to lead to a suspicion of either malady.* But although such an explanation may apply in a few exceptional cases it is obviously inapplicable to such results as those obtained by Mr. Waring, who states that of 300 cases of hepatic abscess proving fatal in India, in only 82 cases, or 27·3 per cent., was the hepatitis preceded by symptoms of dysentery.†

3. The most conclusive cases, however, are those in which the patient has died of hepatic abscess, and no sign of dysenteric ulceration has been found after death. I shall give you immediately the details of a case of this sort, in which, it is important to add,

* Path. Trans. 1862, vol. xiii. p. 120.

† An Enquiry into the Statistics and Pathology of Abscess of the Liver. Trevandrum, 1854.

there had been a considerable amount of diarrhœa during life (Case XXXVII.). Morehead has detailed '17 cases in which there was abscess of the liver without intestinal ulceration,'* while in 204 cases of abscess of the liver collected by Waring there were no ulcerations, cicatrices, or abrasions in 51, or in exactly one-fourth.†

It is clear, therefore, that although dysenteric ulceration of the bowel may occasionally lead to pyæmic deposits in the liver similar to those met with in this country, many cases of tropical abscess are independent of such an origin.

It appears to me that the etiology of hepatic abscess may receive further elucidation from an anatomical point of view. The abscesses of the liver which are met with in this country, and which are the result of purulent absorption, are usually, if not always, small, but numerous. On the other hand, in most cases where abscess of the liver is met with in the tropics, there is but one abscess which attains to large dimensions, or in exceptional cases there may be two or three. Abscesses of the liver, answering to this description, are almost unknown in this climate or in temperate climates generally, except in persons who have sustained some local injury of the liver, or who have at one time resided in the tropics—an extraordinary fact, if its cause be the same as that of multiple abscesses. Accordingly I have proposed to designate this, the *Tropical Abscess*, to distinguish it from the

* Op. cit. ii. p. 12.

† Op. cit.

Pyæmic Abscess, which is the common form in this country.

The experience, however, of Indian practitioners shows that multiple abscesses of the liver are not unknown in the tropics ; but, so far as I have been able to ascertain, this form is never met with except in connection with dysentery, or some other source of purulent absorption. The true tropical abscess may also coexist with dysentery, but from the large number of cases in which both dysentery and hepatic abscess are independent of each other, it follows that when they coexist, they are either the effects of a common cause, which in certain persons will produce either of the diseases separately, or of a concurrence of causes which individually will cause only one of the diseases. Supposing, for example, what is probably the truth, that dysentery is the result of malaria or of contaminated water, and that hepatitis may be caused by a chill in a person whose liver has been congested by residence in a hot climate, aided by irritating ingesta and exposure to malaria (see p. 124), it is readily conceivable that in a country like India where these causes so often operate simultaneously, attacks of dysentery and hepatitis—combined as well as separate—should not be uncommon.

The distinction drawn above between pyæmic and tropical abscess is far from being one merely of pathological curiosity ; it has a most important bearing both on prognosis and treatment. The pyæmic abscess is much the more serious and fatal malady of the

two ; recovery from it, rarely, if ever, occurs. The tropical abscess again is not unfrequently recovered from, and it may burst into the pleura, the lung, the peritoneum, the stomach, the bowel, or externally—accidents to which the pyæmic abscesses are not liable ; and lastly, one of these natural modes of termination of the tropical abscess may be occasionally advantageously imitated by the surgeon, who evacuates the abscess by an external opening—a procedure which would obviously be worse than useless in the pyæmic abscess. It follows therefore that it is of considerable practical importance to be able to distinguish during life between the pyæmic and the form of tropical abscess. The characters of the former have been already detailed ; those of the latter remain to be considered. They are as follows :—

A. In the early stage of the disease, the main clinical features are those of hepatic congestion already described (see p. 120). There is pyrexia ushered in with chilliness, often of a remittent character, and accompanied by pain and tenderness, or a feeling of weight and uneasiness in the region of the liver, and occasionally by pain in the right shoulder, defective respiratory movement of the right ribs, a uniformly augmented area of hepatic dulness, and slight jaundice. The enlargement, however, is on the whole less, and the jaundice of rarer occurrence, than in the congestion of the liver resulting from disease of the heart or lungs. This is due to the circumstance that the branches of the portal vein,

which are gorged in the latter case, are much larger than those of the hepatic artery, which are the main seat of the congestion that precedes the formation of abscess.

B. When the inflammation goes on to suppuration, which, unless it terminate previously by resolution, usually occurs at the end of a week or twelve days, the characters are as follows :—

1. There is enlargement of the liver, which is no longer uniform. The natural outline of the area of hepatic dulness is altered, and will bulge upwards, downwards, forwards, or outwards, according to the direction which the abscess takes in each case (see fig. 14, page 181). Not unfrequently there is a bulging of the ribs, with obliteration of the intercostal spaces, or there is a prominence in the epigastrium, or in the right hypochondrium, such as occurs in hydatid tumours.

2. This bulging or tumour is tense, rounded, smooth, and free from any inequalities. In the advanced stage, however, of exceptional cases, the margin of the enlarged liver may be nodulated from the development in it of small secondary pyæmic abscesses.

3. Fluctuation can usually be detected in the tumour, which will be more or less distinct according to the distance of the abscess from the surface. The feeling of vibration, however, which can often be appreciated on tapping with the finger over an hydatid tumour (page 55), cannot be elicited in an abscess, owing to the greater thickness of its con-

tents. Another distinctive character of abscess is, that the fluctuation is usually surrounded by a mass of inflammatory hardness.

4. Pain and tenderness are present in most cases. The pain, however, is dull and heavy, and is rarely of that acute character, in the first instance, at all events, so common in the pyæmic abscess. This is because the abscess is usually at first in the interior of the liver. The pain only becomes acute, and the tenderness great, when the matter approaches the surface and excites peri-hepatitis, or stretches the integuments. Some cases are remarkably latent, as far as pain is concerned, throughout their whole course, while in others pain is only produced when the patient takes a long breath, and at the same time pressure is made below the margin of the ribs. A sympathetic pain in the right shoulder is not uncommon, when the abscess is situated on the convex surface of the right lobe; but in the majority of cases it is absent. The presence of pain in the shoulder will undoubtedly increase, although its absence will not diminish, the importance of other symptoms.

5. Ascites, œdema of the lower extremities, enlargement of the superficial veins of the abdomen, and hæmorrhoids are not distinguishing characters of tropical abscess, any more than of hydatid of the liver. Their occurrence in rare cases is accidental, and due to compression by the tumour of the trunk of the portal vein or of the inferior vena cava. Oc-

asionally fluid is thrown out into the peritoneum as the result of peritonitis.

6. Enlargement of the spleen is rarely present in tropical abscess.

7. Jaundice is a much rarer symptom in the tropical than in the pyæmic abscess. Its occurrence, in fact, if we except a slight icteric tint during the primary stage of congestion, is quite exceptional. Morehead has noted it in only five out of upwards of 120 cases.* When it occurs, it has mostly a mechanical origin, and is due to the concurrence of catarrh of the bile-ducts, or to the direct compression of the large ducts by the abscess.

8. The constitutional symptoms are important, as serving to distinguish the tropical abscess from hydatid tumour. After the occurrence of suppuration, they are mainly progressive emaciation and fever of the hectic type. Rigors and night-sweats, however, are less prominent than in the pyæmic abscess. The tongue in many cases is preternaturally red, and more or less coated with aphthæ; diarrhœa is not uncommon; the urine is high-coloured, and throws down a copious sediment of lithates or lithic acid; and often there is a short dry cough.

9. The duration of tropical abscess of the liver is a matter of some importance in diagnosis. Whether fatal or not, it rarely lasts more than six months after the occurrence of obvious swelling, yet on the whole the course of the disease is less rapid than that of the pyæmic abscess. Cases, too, have been known

* Res. on Dis. in India, 1860, p. 373.

where a small tropical abscess with organized walls has existed for months, or even perhaps for years, in a quiescent form, and has then undergone enlargement and burst. Some of the cases met with in this country, where a large abscess forms in the livers of persons years after their return from India, admit perhaps of this explanation.

10. The circumstances under which 'tropical abscess' occurs, its frequency in the tropics, and its extreme rarity in temperate climates, except in persons who have visited the tropics, may sometimes be of material assistance in its diagnosis.

11. The diseases most likely to be confounded with tropical abscess are hydatid tumour, inflammatory enlargement of the gall-bladder, pyæmic abscesses, and abscess of the abdominal parietes (see p. 20).

a. An hydatid of the liver is the enlargement most likely to be mistaken for abscess. In both there is an *unequal* enlargement, presenting fluctuation and occasionally causing bulging of the ribs or a semi-globular tumour in the epigastrium. Tropical abscess is mainly to be distinguished from hydatid by the presence of pain, by its much more rapid course, and by its constitutional symptoms. The possibility, however, already referred to, of an hydatid tumour suppurating, or becoming converted into an abscess, must not be lost sight of. An error in diagnosis from this cause is all the more likely to arise if the patient, as often happens, has been ignorant of the existence of the hydatid tumour prior to the occurrence of the

acute symptoms due to its taking on inflammatory action.

b. The circumstances under which enlargement of the gall-bladder may simulate hepatic abscess and its distinguishing characters will be considered in a subsequent lecture.

c. The constitutional symptoms of tropical and pyæmic abscesses may be identical. For distinguishing them, we must rely mainly on the form of the enlargement and the circumstances under which it occurs (see pp. 150 and 171).

Treatment.—A. *Before suppuration* has occurred, the rules for treatment will differ but little from those already laid down for congestion of the liver. But acute congestion in tropical climates is often benefited by more active measures than would be justifiable in the forms of congestion which are more common in this country.

1. When the disease sets in suddenly, and when the pulse is full and firm and the temperature high, a full bleeding from the arm, by diminishing the force of the heart and the total amount of blood, will sometimes appear to check the advance of the disease to suppuration. This remark applies especially to Europeans who have not resided long in the tropics, and who have not suffered from the injurious effects of malaria, intemperance, &c.

2. Local depletion by means of leeches may be had recourse to after general bleeding, or independently, in those cases where general bleeding is unsuitable.

3. Poultices and warm fomentations to the region of the liver often give great relief, and should be employed in all cases, whether depletion be practised or not.

4. In those cases where the symptoms of congestion pass into a chronic state, but at the same time there is no evidence of suppuration, a small blister often appears to do good.

5. Saline purgatives, with occasional doses of calomel or blue pill, alkalies, diaphoretics and diuretics, must be employed according to the rules already laid down (see p. 126).

6. The diet must be of that bland and restricted character which I have already told you is essential to the proper treatment of ordinary congestion (see p. 125).

B. In tropical abscess of the liver, not only may we hope to prevent suppuration by appropriate treatment, but even *after* it has occurred, the case is far from being, as in the pyæmic abscess, necessarily fatal. The treatment, however, for the stage antecedent to suppuration is no longer suitable.

1. Warm fomentations and poultices are still to be applied to the region of the liver; and in the event of acute pain supervening, a few leeches will often give relief.

2. The patient's strength must be supported by mineral acids and vegetable tonics, and in particular by the sulphuric or nitric acid with quinine.

3. Opium is in most cases necessary to relieve

pain, to procure sleep, or to allay the harassing cough.

4. Purgatives are no longer called for. If the bowels be constipated, a mild laxative may be given from time to time, but more commonly there is diarrhœa or dysentery, necessitating the use of vegetable and mineral astringents, with opiate enemata or suppositories. Mercury in this stage is worse than useless, and is now almost universally discarded.

5. The diet must be of a much more generous nature than that which is permissible in the stage of congestion; and when the circulation is weak, small quantities of wine or brandy will be necessary.

6. In multiple abscesses it is clear that no advantage is to be derived from operative interference, but when there is a single large abscess, the propriety of evacuating the pus may fairly be entertained. It is no doubt true that a large abscess of the liver may become encysted and shrivel up, and in this way undergo what may be called a spontaneous cure, independently of rupture, but this is an event so rare that it cannot be calculated on. Recovery also takes place occasionally in consequence of the abscess emptying itself through a bronchial tube, into the bowel, or externally through the abdominal parietes; but the process is tedious, and even when it occurs many patients die of exhaustion from fever, pneumonia or diarrhœa, to say nothing of their liability to destruction at any moment from the abscess bursting into the pericardium, the pleura, or the peritoneum.

In a large proportion of cases, however, the patient dies while the abscess is still confined to the liver.* Under these circumstances the expediency of hastening the evacuation of the matter naturally suggests itself.

You will find nevertheless that professional opinion is divided on this important question. Dr. Budd, in his standard work on 'Diseases of the Liver,' considers the dangers of operating so many and so great that it is better to let matters alone and allow the abscess to open of itself; † and more recently a similar opinion has been strongly expressed by Professor Maclean, of the Army Medical School at Netley. ‡ Some authorities, again, such as Frerichs § and Morehead, || advocate opening the abscess in selected cases; while others, like Dr. Murray, formerly Inspector-General of Hospitals in Bengal, Dr. Cameron, ¶ and Sir Ranald Martin,** maintain that 'when we have just grounds for believing that abscess of the liver exists, we ought not to lose a day in evacuating

* Of 300 fatal cases of hepatic abscess collected by Mr. Waring, the abscess, at the time of death, had not extended beyond the boundaries of the liver in 169; in 48 it had been opened by operation; in 42 it had opened spontaneously into the right lung or thoracic cavity; in 15, into the peritoneum; in 8, into the stomach or colon; in 3, into the hepatic vein, &c. (An Enquiry into the Statistics and Pathology of some points connected with Abscess in the Liver, as met with in the East Indies. Trevandrum, 1854.)

† Op. cit. 3rd ed. 1857, p. 124.

‡ Lancet, July 18, 1863.

§ Dis. of Liver, Syd. Soc. Ed. ii. p. 147.

|| Res. on Dis. in India, 2nd ed. 1860, p. 410.

¶ Lancet, June 6 and 13, August 8, 1863.

** Lancet, August 20 and 27, 1864.

it by puncture, and that we are both justified and safe in endeavouring to hit upon it with a trocar when deep-seated, avoiding the gall-bladder and large veins.* Dr. Cameron, in fact, goes so far as to recommend exploring the liver with a trocar, in cases where the existence of an abscess is suspected though not certain, and has published cases where no pus was found, and yet the patient's symptoms subsided instead of being aggravated subsequently to the exploration. Amid such conflicting opinions we may be aided in forming a just judgment by considering, on the one hand, the dangers of the operation, and, on the other, the dangers of non-interference.

The main objections raised against the operation are as follows :—

a. That pus is apt to escape into the peritoneum and excite fatal peritonitis. In most cases, however, when the abscess is near the surface, there would be adhesions which would prevent the entrance of pus into the peritoneum. Morehead speaks of the absence of adhesions as quite exceptional (in only 3 of 76 fatal cases). Moreover, if desirable, it is always possible to produce adhesions.

b. That air will enter the abscess and excite fresh inflammation. This is an undoubted source of danger ; but it is as (if not more) likely to be incurred if the abscess opens spontaneously into the bowel, a bronchial tube, or externally.

c. That the mechanical injury of the puncture is

* Cameron. *Lancet*, June 6, 1863, p. 631.

apt to produce hæmorrhage and fresh inflammation in the hepatic tissue. So far as I have been able to ascertain, this is an objection founded on theoretical considerations, rather than on actual observation. I have had several opportunities of confirming Dr. Cameron's statement to the effect that a fine trocar can be plunged into the liver without any ill result except a little local pain, and without, in fact, any trace of the puncture being discernible when death occurs shortly afterwards.

d. That the fatal event may be hastened by gangrene of the tissues around the wound.* This accident has been chiefly observed when the opening has been made in an intercostal space, and then, as Morehead has shown, it occurs alike when a spontaneous rupture takes place, and when a puncture is made.† The gangrene is most probably connected with the caries or necrosis of the ribs, which is almost always present in these cases, and which would probably not occur were the abscess opened before the ribs became implicated.

The chief dangers of non-interference are these :—

a. The abscess becomes daily larger, more and more of the hepatic tissue is destroyed, and ultimately the gland may be reduced to a mere sac containing pus, while adjacent organs are compressed and the ribs are eroded.

* Maclean. *Lancet*, July 18, 1863.

† *Op. cit.* p. 410.

b. The patient may die suddenly from the abscess bursting in various directions.

c. The great majority of patients with abscess of the liver die of exhaustion from hectic fever or diarrhoea, either while the abscess is still confined to the liver, or after it has burst.

Statistics have been appealed to with the object of proving the uselessness of operative interference. Of 81 cases where the abscess was opened, collected by Mr. Waring, only 15 (or 18·5 per cent.) recovered, and of 24 cases recorded by Morehead, only 8, or one-third, recovered. But in many of these cases death was due, not to the operation, but probably to this having been too long delayed; while several of Waring's cases were examples of multiple abscesses, for which an operation is obviously unsuited. Moreover, of 203 cases collected by Rouis, where the abscess was not opened, 162 (or 80 per cent.) died.*

After duly balancing, then, the dangers of operation against the dangers of expectancy, I do not hesitate to recommend to you the propriety of evacuating the pus in a large number of cases of tropical abscess of the liver. The operation may not be free from danger, but to wait in these cases upon Nature, as it is called, is to wait upon Death, and I would suggest for your guidance the following rules:—

a. In all cases where there is a visible fluctuating tumour, operate at once.

* Frerichs, Op. cit. ii. p. 136.

b. In cases where the symptoms of abscess of the liver are present, with a distinct tumour projecting from the normal contour of the liver; or causing bulging of the ribs, even though there be no perceptible fluctuation, it will be well to operate.

c. When symptoms of abscess coexist with uniform enlargement of the liver, but with no distinct tumour or bulging, if there be any local œdema, or obliteration of an intercostal space, or acute pain, always localized to one particular spot when the patient takes a full inspiration, it will be well to operate; but if there be no such œdema or obliteration or pain, it may be better to wait, as the enlargement may possibly be due to multiple abscesses, or if there be but one abscess, it is doubtful if it will be reached.

When the operation is resolved on, it may be performed as follows:—

a. When there is distinct pointing with an inflammatory blush of the skin, an opening may be made with a bistoury.

b. Under other circumstances, a small trocar will be preferable, and it ought to be introduced wherever there is the slightest fulness or superficial œdema, or acute pain.

c. When the abscess is small, not holding more than ten or twelve ounces, it ought to be completely evacuated, and the canula tied in for two or three days. On its removal, a tent of lint dipped in oil may be substituted.

d. When the abscess is very large it will be better

to evacuate it by instalments at short intervals, carefully excluding the air on each occasion.

e. In the exceptional cases, where no adhesions exist, it will be prudent to produce them by the local application of caustic potash, before puncturing.

f. After the operation, a large warm poultice should be applied over the liver, and the patient should lie on it, taking care that, if the canula has been left in, pressure upon it is obviated by a suitable pad or pillow. A full dose of morphia ought also to be administered at once.

The first of the following cases is an excellent illustration of tropical abscess of the liver independent of dysentery, notwithstanding that after the formation of pus, diarrhoea was a prominent symptom. It is a matter of regret that the abscess was not punctured; but fifteen years ago this operation was rarely practised.

CASE XXXVII.—*Tropical Abscess of the Liver—No Dysenteric Ulceration of the Bowel.*

Private H. C——, aged 33, of the 2nd European Bengal Fusiliers, was admitted under my care into the Military Hospital at Prome, on November 12, 1853. His habits had been very dissipated; he had suffered from many attacks of fever and congestion of the liver, and shortly before his admission he had been exposed almost continuously for three weeks to wet on the decks of steamers during the passage from Calcutta to Rangoon and from Rangoon up the Irrawaddi to Prome. He had never had dysentery. He began to suffer from febrile symptoms and pain in the right side in the first week of October during the passage from Calcutta, but his condition did

not prevent him attending to his duty until a few days before admission, when the pain in the side became much more severe.

On admission, the patient's pulse was 112, and his skin hot. He complained of much pain in the region of the liver, and stretching from that up to the right shoulder. The pain was greatly increased by coughing or taking a long breath, and there was considerable tenderness on pressure over the epigastrium and below the right ribs. The hepatic dulness in the right mammary line measured 6 inches. Posteriorly and upwards the margins of hepatic dulness were normal, and the increased size appeared due to a bulging from the lower margin. There was no fluctuation and no jaundice or ascites, but there was less movement of the ribs in respiration

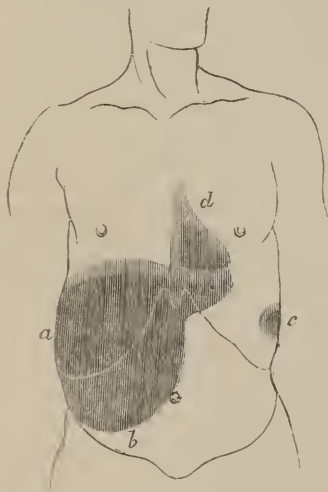


Fig. 14 represents the Outline of Hepatic Dulness, and the Bulging of the Ribs (*a*) in H. C—, on December 2, 1853.

on the right side than on the left, and frequent cough. The tongue was moist and coated white; there was frequent vomiting; a day or two before admission the bowels had been relaxed, but at the time of admission they were costive. There was some scalding in micturition; the urine was high-coloured; sp. gr. 1027; it contained no albumen, but deposited crystals of lithic acid.

The patient was cupped to 8 ounces over the liver, and during the first week after admission was treated with calomel and opium, and subsequently with nitro-muriatic acid, quinine, opiates, and wine.

On November 18, diarrhœa came on, with profuse night-sweats, but no rigors. The vomiting continued, and the tongue was clean, very red, and deeply fissured. The cough and scalding in micturition had abated, but the vomiting and diarrhœa persisted, notwithstanding the use of remedies. On November 20, the tongue was noted as dry and brown, and the patient became very emaciated, but was comparatively free from pain, until November 26, when he was seized with acute pain, shooting up from the region of the liver to the right shoulder. On the following day this had subsided; and after this there was but little vomiting or purging, and the symptoms were mainly those of hectic fever, with increasing prostration, until December 1, when there was noticed below the right ribs, rather to the right of the mammary line, a distinct smooth rounded bulging, with obscure fluctuation in the centre. The hepatic dulness in the right mammary line was now 8 inches, the increase being due to a projection downwards from the lower margin of the normal area of hepatic dulness. There was also considerable bulging of the lower right ribs. The patient was now free from pain, the vomiting and purging had ceased, but the cheeks were sunken and presented a hectic flush, the fever and night-sweats continued, the tongue was dry and brown, and the teeth coated with sordes.

On December 8, the patient was in a state of extreme prostration; on the following day his mind was wandering, and at 9.30 P.M. he died.

On examination of the body ten hours after death, one enormous abscess was found in the right lobe of the liver. It contained upwards of four quarts of pus, having a reddish tint, and under the microscope presenting numerous pus-corpuscles, with oil-globules and hepatic cells undergoing disintegration. The walls of the abscess were formed by ragged masses of hepatic tissue coated with inflammatory products; at two places the walls were very thin, one situated below the margin of the right ribs, and corresponding to the tumour observed during life, and the other posteriorly near the mesial fissure. The stomach and the small and large intestines presented no trace of cicatrices or of recent ulceration. The spleen, lungs, and heart were normal. There were old

adhesions between the opposed surfaces of the left pleura; and the cavities of the heart, but particularly the right, contained large masses of decolorized fibrine.

In the next case, there was ulceration of the colon, but this appeared, from the history and post-mortem appearances, to be secondary to the abscess of the liver. The specimen was exhibited by me to the Pathological Society, and the case is recorded in the 8th volume of the 'Transactions.'

CASE XXXVIII.—*Large Abscess of the Liver opening into the Ascending Colon.*

J. P.—, a man aged 40, was admitted into St. Mary's Hospital, under the care of Dr. Sibson, on April 18, 1856.

He stated that he had always enjoyed good health and that, although he had been in the habit of drinking a good deal of malt liquor, he had never been addicted to spirits, and had, on the whole, been a temperate liver. He had never been abroad. About a month before admission, he 'took cold,' and was seized with a shooting pain in the right hypochondriac region, which on the second day became so extreme as to prevent his working. He went to bed, where he remained until the day of admission, the pain in the right side continuing without intermission except when relieved by opium.

On examination after admission, there was found to be a great fulness in the right hypochondriac and lumbar regions of the abdomen, with a feeling of a resisting mass extending downwards as far as the crest of the ilium, and forwards to within 3 inches of the linea alba. This space was universally dull on percussion, and the dulness was continuous with that of the liver. The upper margin of the area of hepatic dulness was not elevated, and the dimensions of the left lobe appeared normal. The swelling was of a doughy consistence, and presented indistinct fluctuation. The tongue was loaded, the bowels rather confined. The urine was voided three or four times a day and was acid; sp. gr. 1020. The pulse was 108, weak.

Poultices of linseed meal were applied over the swelling, while iodide of potassium (gr. ij ter die), gentle laxatives, opiates, and stimulants were prescribed internally.

On April 24, he had an attack of erysipelas of the face, which continued for four or five days. On April 26, during this erysipelas, he was seized with violent diarrhoea. This ceased in a great measure after four or five days, and he then felt himself greatly better; his appetite had improved, the pain had gone, the swelling and dulness were much diminished, and the calls to make water less frequent. He continued to improve until May 11, on which day he had a return of the severe pain and diarrhoea, with purulent stools. The pain was referred chiefly to a spot about two inches below the margin of the ribs, in a line with the right nipple. The stools were of a light buff colour and very offensive. This diarrhoea resisted all treatment, and soon the patient's constitution began to give way. He became liable to febrile exacerbations towards evening, and profuse perspirations during the night. His pulse varied from 100 to 125, and was very weak, his tongue became dry and brown, and he gradually sank until death at 10 P.M. on May 27. Four days before death the swelling in the right side was observed to have very greatly diminished, the dulness in the right lumbar region not extending farther forward than a perpendicular line drawn from the middle of the crest of the ilium to the ribs.

Post-mortem examination forty-one hours after death.—On opening the abdomen there were found extensive adhesions of the viscera and other indications of peritonitis, which were, however, entirely limited to the right side, the peritoneum on the left side being perfectly normal. These adhesions of the viscera on the right side rendered their examination extremely difficult. The whole of the anterior margin of the right lobe of the liver was firmly adherent to the peritoneal surface of the abdominal wall, while the under surface of the anterior edge, along with the gall-bladder, was in intimate union with the transverse colon. The texture of the liver was pale. In the lower part of the right lobe was an abscess as large as two fists, containing a quantity of fluid fæculent matter, of a light yellow colour. This abscess involved almost the whole of that portion of the lobe to the right of the fissure of the gall-bladder, and extended to within half an inch of its upper surface. The upper two-thirds of the walls of the abscess were formed by hepatic tissue, and were rough and ragged, without any limiting membrane. The lower part was completed by the kidney, the anterior layer of the fascia lumborum, and about 3 inches of the

ascending and transverse colon. This portion of the colon communicated freely with the cavity of the abscess. Its upper wall next the abscess presented a cribriform appearance, all that remained of it being a few narrow bridges, passing transversely, and easily torn across. There was extensive ulceration of the adjacent portion of the ascending colon, and slight ulceration of Peyer's patches in the ileum.

The kidneys were anæmic, and the spleen soft and friable. The thoracic organs were healthy, the left cavities of the heart containing blood, the right being empty.

In Case XXXIX. the patient had suffered from dysentery, and died from the bursting of the abscess upwards through the diaphragm.

CASE XXXIX.—*Abscess of the Liver opening upwards through the Diaphragm—Secondary Abscess of Liver.*

I show you here a specimen which I removed some years ago from the body of a patient—a man, aged 34, who died in this hospital, and which illustrates the bursting of a large abscess upwards through the diaphragm. In this case, the patient had suffered some years before from dysentery, in India and Malta. His symptoms during the nine days that he was in the hospital before his death were hectic fever and emaciation, dyspnoea, cough, and purulent expectoration, with a painful enlargement of the liver, producing an outward bulging of the ribs. The hepatic dulness extended only 2 inches below the margin of the ribs in the right mammary line, but upwards it reached to the third intercostal space. The enlargement felt smooth, but did not involve the whole organ uniformly. The tongue was unusually red; there was no vomiting, jaundice, or diarrhoea, but the abdomen generally was tender, and there was distinct evidence of fluid in the peritoneum.

After death, three or four pints of flaky serum were found in the peritoneum. The liver was firmly adherent to the diaphragm and abdominal parietes, and in the upperpart of the right lobe was an abscess as large as a cocoa-nut, which had perforated the diaphragm so as to be bounded above by the base of the right lung. The abscess was enclosed in a dense capsule of areolar tissue, and

contained yellow pus with large fibrinous flakes. In the lower lobe of the right lung was another abscess, the size of a large orange, distinct from the former, and containing pinkish pus. The descending colon and sigmoid flexure were much contracted; their coats were thickened; the mucous membrane was of a slaty hue, but presented no recent ulcers or distinct cicatrices.

The last case which I shall mention is a good illustration of the benefit which may often be derived from evacuation of the abscess.

CASE XL.—*Tropical Abscess of the Liver—Puncture with a Large Trocar—Recovery.*

Mr. C. D.—, aged 23, consulted me on June 11, 1867. He had arrived from Calcutta the day before, and gave the following account of himself. He had resided in Calcutta for about three years, and had lived freely, but had never suffered from dysentery. He had been taken ill about the end of March with febrile symptoms, and rapidly increasing prostration. He had no pain in the side, no diarrhoea, and no jaundice, but about April 12 a tumour made its appearance below the right ribs, which rapidly increased until the 19th, when it was opened with a large trocar, and upwards of a pint of matter let out. The canula was left in the wound, and on the 21st the patient was put on board the overland steamer in so prostrate a state that he was hardly expected to recover. He slowly improved, however, during the voyage, and the canula was removed at Aden about a fortnight afterwards. I found an opening with pouting granulations about half-way between the umbilicus and the ribs, and 2 inches to the right of the median line, from which about two drachms of thin pus escaped daily. The patient was weak and anæmic, but in other respects appeared to have nothing amiss. He was treated with mineral acids, quinine, and iron, and within three months he had regained his usual health and strength. There was no evidence of enlargement of the liver, and the opening had permanently closed. (With the exception of an attack of gout in January, which he had previously suffered from, and of which disease his father had died, he remained in good health until he returned to India in February 1868.)

LECTURE VI.

ENLARGEMENTS OF THE LIVER.

CANCER.

TUBERCULAR ENLARGEMENT—LYMPHATIC ENLARGEMENT—MULTI-
LOCULAR HYDATIDS—ENLARGEMENTS OF GALL-BLADDER.

THE last form of enlargement of the liver, the clinical characters and treatment of which have to be considered, is that which is due to cancerous deposit.

X. CANCER OF THE LIVER.

Cancer of the liver may be recognized by the following clinical characters.

1. The size of the liver is increased, and not uncommonly the enlargement is very great, so that the organ fills a great part of the abdominal cavity. A cancerous liver has been known to weigh 250 ounces, or about five times the normal weight.* The enlargement is progressive, and in the softer forms of cancer may be so rapid that a weekly increase may be noted. On the other hand, it must be remembered that the

* See Budd, Dis. of Liv. 3rd ed. p. 407, and Path. Trans. xviii. p. 145.

liver may contain a considerable amount of cancer, and yet the enlargement may not be appreciable during life. The liver may have been originally a small one, and the addition of the cancer may not cause it to project beyond the costal arch, or the lower margin may be overlapped by a distended bowel. You will remember the case of Mary T——, a very fat woman, 54 years of age, who died recently in the hospital, of apoplexy supervening upon white softening of the brain (with hemiplegia), and whose liver was unexpectedly found studded with large cancerous nodules, although the organ did not project beyond the costal arch, and there had been no symptoms during life of disease of the liver. A similar observation was made in two other cases, which I shall detail to you immediately (Cases XLVI. and XLVII.).

2. The enlargement is usually irregular, from the presence of nodular cancerous excrescences projecting from the surface or from the margin of the liver, which can often be felt on palpation, and are sometimes even visible through the abdominal parietes. Occasionally the cancerous deposit forms one large excrescence or tumour at a particular portion of the organ. Dr. Bright has recorded some remarkable cases in which the tumour was confined to the left lobe, and projected downwards into the abdomen, or upwards into the left side of the chest;* and the specimen I show you here, obtained from the body of

* Abdom. Tumours, Syd. Soc. Ed. pp. 261 and 308.

a patient who died under my care in the Fever Hospital, is another illustration of the same condition (Case XLV.). More commonly a number of nodulated outgrowths, about the size of cherries or small oranges, project from the portion of liver which is

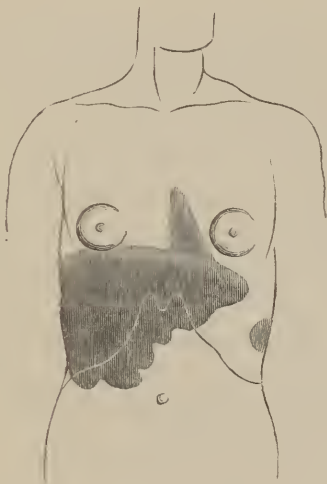


Fig. 15 shows Area of Hepatic Dulness, in Hannah C—— (Case XLI.), with nodulated lower margin.

opposed to the abdominal parietes (see fig. 15). Care must be taken not to mistake for such excrescences the rigid bellies of the recti muscles (see page 20). It is necessary also to remember that a nodular character is not essential, as might be inferred from some descriptions, to cancerous enlargement of the liver. In certain cases the cancer is not

deposited in the liver in isolated nodules, but is infiltrated through the hepatic tissue in such a way that, although the organ may be greatly enlarged, its natural outline is but little altered; and even in the nodular form of cancer, the portion of liver below the ribs is sometimes quite smooth (Case XLII.).

3. The enlargement feels very hard and resistant on palpation, and nowhere exhibits any fluctuation. In very rare cases some of the cancerous nodules may be so softened as to present obscure fluctuation.

4. A cancerous liver is almost always painful and tender on pressure, and very often the pain radiates to the shoulder, back, and loins. At first there may only be a feeling of weight and uneasiness in the right hypochondrium; but after a time the pain is often lancinating, and the tenderness acute, and both are particularly severe in cases where the growth is rapid, or where, as often happens, there is inflammation of the superimposed peritoneum.

5. Jaundice is present in a large number of cases, and when once developed it never disappears. The coexistence of enlargement of the liver with persistent jaundice ought always to raise the suspicion of cancer. The jaundice usually results from the compression of the bile-duct by a cancerous nodule in the liver or by enlarged glands in the portal fissure. If the ducts be not thus compressed, almost the whole of the secreting tissue may be destroyed without any jaundice resulting. Of 91 cases of cancer of the liver collected by Frerichs, 52 died without ever having been jaundiced.

6. Fluid in the peritoneum is observed in more than one-half of the cases of cancer of the liver, before the fatal result. It may concur with jaundice, or each may exist independently (see Cases XLI. XLII. and XLIII.). The fluid may be a simple dropsical collection, due to compression or obstruction with cancerous matter of the trunk or large branches of the portal vein, but the amount is usually small as compared with what is observed in cirrhosis. Oftener it is the result of a chronic peritonitis originating on the surface of the liver. Now and then, as in Cases XLIV. and XLV., blood is thrown out into the peritoneum from a rupture in a fungating or softened cancerous mass in the liver.

7. The superficial abdominal veins are only enlarged in those comparatively rare cases where the portal circulation is seriously obstructed.

8. Enlargement of the spleen is rare, and this constitutes an important distinction of the cancerous from the waxy or cirrhotic liver.

9. The constitutional symptoms, in the first place, are mainly those of deranged digestion, such as nausea, flatulence, and constipation, and occasionally attacks of vomiting or diarrhoea. A short dry cough is not uncommon. When the cancer grows rapidly, there is often a certain amount of pyrexia, with a furred tongue, and scanty high-coloured urine throwing down a copious deposit of lithites. Before the disease has lasted long, the patient presents in a marked degree the phenomena of the cancerous cachexia—extreme anæmia, with an earthy chlorotic

colour of the integuments (unless there be jaundice), and rapidly increasing debility and emaciation. These symptoms are always aggravated by the co-existence of cancer of the stomach.

10. Cancer of the liver is, in most cases (fully three-fourths), secondary to cancer of some other organ, such as the stomach, uterus, the female breast, the rectum, or the vertebræ. In more than one-third of the cases it is secondary to cancer of the stomach. The symptoms of cancer in these various organs will therefore materially aid the diagnosis. Even when the cancer is deposited first in the liver, other parts, such as the cœliac, mediastinal and cervical glands, and the lungs, are apt to become affected, and thus throw fresh light on the primary disease (see Case XLIII.).

11. Cancer of the liver always runs a rapid course. The medullary cancer often grows very rapidly, and is fatal within a few weeks or months; and although scirrhus is said sometimes to last for two years,* it is rarely prolonged beyond twelve months. The very fact of an enlargement of the liver having lasted much longer than this, would be an argument against its being due to cancer.

12. The age of the patient is sometimes of assistance in diagnosis. Cases are extremely rare where the liver is primarily affected with cancer before 35 or 40. Secondary cancer of the liver, it is true, may occur at any age, but then the primary disease will point to the nature of the case.

* Budd, Dis. of Liver, 3rd ed. p. 413.

13. The mistakes most likely to occur in diagnosis consist in mistaking cancer of the liver for waxy disease or cirrhosis. (See also p. 61.)

a. The smooth infiltrated form of cancer may be mistaken for waxy degeneration. In both there is a smooth, uniform, very hard enlargement of the liver; but in the waxy enlargement the progress of the disease is slow, there is an absence of pain or of the cancerous cachexia, and there is usually also enlargement of the spleen, with albuminuria, and a history of constitutional syphilis, caries of bone, or protracted discharge from a suppurating surface; whereas in cancer there is no enlargement of the spleen or albuminuria, but the course of the disease is rapid, and there are pain, cachexia, and often signs of cancer elsewhere.

b. More rare cases, where cirrhosis and waxy disease coexist (see pp. 26 and 42), may be mistaken for nodular cancer. In both there may be a nodulated hard enlargement of the liver with ascites. The points of distinction are the same as between the smooth forms of waxy disease and cancer.

Treatment.—The treatment of cancer of the liver must be entirely palliative. There is no known remedy which can arrest or retard its progress. Mercury, iodine, arsenic, and the *Sanguinaria Canadensis*, which at different times have been recommended for the purpose, have been shown to be worse than useless. In none of the many diseases of the liver for which it has been the fashion to give mercury,

has it been productive of so much injury as in cancer. The treatment must be restricted to supporting the patient's strength and nutrition by appropriate food, correcting errors in digestion, relieving pain, and procuring sleep.

1. The diet ought to be nutritious, but digestible, and ought to contain a large proportion of the nitrogenous principles of food, and comparatively little of saccharine and oily substances, which are calculated to increase the work thrown upon the liver. Alcoholic stimulants will often be necessary in the advanced stages of the disease, but ought to be given in moderation and well-diluted. In those hopeless cases where the primary disease is cancer of the stomach, the diet must consist mainly of milk and animal soups.

2. Various remedies will often be necessary to correct errors in digestion. For vomiting, bismuth, hydrocyanic acid, lime-water, creosote, or ice, will be useful, and likewise the occasional application to the epigastrium of a sinapism or small blister. In the latter case, great advantage is often derived from sprinkling over the blistered surface a quarter of a grain of morphia. The use of blisters for any other object can do no good, and can only weaken the patient and put him to unnecessary pain. Flatulence will be relieved by the ethers and essential oils, but better still by such remedies as charcoal, creosote, or carbolic acid, which absorb the gas, or, by arresting decomposition, prevent its formation. A dose of from fifteen to thirty minims of a saturated

aqueous solution of carbolic acid, with a few drops of chloric ether in peppermint water, is often a most effectual remedy for this symptom. The bowels are often constipated, and will require relief, but care must be taken to avoid castor-oil and powerful purgatives, which will either nauseate the stomach, or lower the patient by producing copious watery discharges. Four or five grains of the compound rhubarb pill with a grain of blue pill and a grain of extract of henbane, will usually produce the desired result satisfactorily and safely, or the bowels may be cleared out from time to time by a simple enema.

3. Sooner or later, in most cases, anodynes will be necessary to relieve pain or procure sleep. Belladonna conium, or Indian hemp, will often be found useful for these objects, and ought to receive a trial in the first instance; but in most cases it will be necessary to have recourse ultimately to one of the various preparations of opium or morphia. The solution of the bimeconate of morphia, which is of the same strength as laudanum, has less tendency to derange the stomach or constipate the bowels than most other forms in which opium is given; and these disadvantages of opium will also be, in a great measure, avoided by the subcutaneous injection of morphia. Lastly, poultices and warm fomentations, with or without a few leeches, may be required for intercurrent attacks of peri-hepatitis.

The following cases, which, with two exceptions,

have been under your observation, illustrate the remarks that have now been made on cancer of the liver.

CASE XLI.—*Cancer of the Liver and Ovary—Jaundice, but no Ascites.*

Hannah C——, aged 50, a cook of large build and rather stout, married, and mother of one child, was admitted into the Middlesex Hospital under my care on July 28, 1863. She stated that for many years she had been subject to ‘bilious attacks’ (vomiting and headache), but that about two years before admission, these attacks became much less frequent and severe, and she had enjoyed good health, until about ten weeks before admission, when she was attacked somewhat suddenly with pain in the epigastrium and right hypochondrium and in both shoulders, accompanied by great languor, and followed next day by diarrhœa, which lasted a week. A month before admission, the pain became much increased, and the urine was noticed to be of a dark greenish-brown colour; a week later the skin became yellow, and since then, the patient has suffered much from itchiness. From the first she had been losing flesh.

The symptoms, while the patient was under my observation, were as follows :—The skin, conjunctivæ, and serum of a blister were of a bright orange colour, and there was great itchiness of the entire surface. The tongue, at first clean, became afterwards coated with a thin white fur. At first, there was no vomiting, but frequent attacks of nausea and a feeling of distention and oppression after meals. The appetite was very bad. The motions were destitute of any trace of bile; they were pultaceous, clay-coloured, and very offensive. Much pain was complained of in both shoulders, and in the epigastrium and right hypochondrium; this was much greater a few days after admission than subsequently. The liver was much enlarged, the hepatic dulness, in the right mammary line, extending from $\frac{1}{2}$ an inch below the nipple to $1\frac{1}{2}$ inch below the ribs, and measuring $6\frac{1}{4}$ inches. The portion of liver below the ribs was hard, tender, and distinctly nodulated (fig. 15, p. 189). There was no evidence of ascites. The urine was scanty, only about one-half of the normal quantity, sp. gr. 1030, acid, dark like porter, and threw down a copious deposit of lithates. It contained abundance of bile-

pigment, but no bile-acids (by Harley's test), and no albumen. The pulse was 60; the cardiac and respiratory signs were normal, except that occasionally 'crackling sounds' were heard over the base of the right lung. On August 6, and again on August 15, it was noted that the patient vomited after her medicine. On August 29, there was a considerable increase of the pain and tenderness in the abdomen, with vomiting and pinched features. Under treatment, these symptoms abated somewhat, but the vomiting returned from time to time, while the languor and prostration rapidly increased. On September 28, the vomiting became incessant, and from this date the patient gradually sank, until death on October 3.

The treatment consisted in bismuth, hydrocyanic acid, and opiates, sinapisms to epigastrium, and nutritious but digestible food.

Autopsy.—The body was well nourished, and there was a thick layer of fat everywhere beneath the skin, in the omentum, and around the kidneys. The tissues throughout the body were deeply stained with bile. There was no fluid in the peritoneum, and no sign of recent peritonitis. The mucous membrane of the stomach and intestines was normal, but the contents of the bowel contained no trace of bile, and none could be squeezed from the gall-bladder into the duodenum. The liver was very large, weighing 97 oz., and its right lobe, measuring 13 inches from before backwards. Its surface was studded with elevated yellowish white, moderately firm nodules, varying in size from a pea to a walnut, and many of them depressed in their centres. Similar masses were seen in the interior of the liver on section. One mass, the size of a large orange, occupied the entire thickness of the right lobe in front, extending back to the transverse fissure, and in contact with the upper surface of the gall-bladder. These masses yielded a creamy juice on section, which contained characteristic 'cancer-cells;' some of them were softened in the centre into a yellow pulp, and here the cancer-cells contained much oil, and there were many compound granular cells. The hepatic lobules between the cancerous masses had a peculiar appearance. The central third of each lobule had a dark olive-green colour, and the hepatic cells in it contained much bile-pigment; the outer two-thirds were pale-yellow, and there the secreting cells were loaded with oil. Several stellate crystals of tyrosine were found in the secreting tissue. The gall-bladder contained no bile, but was filled with faceted gall-stones. The hepatic ducts were considerably dilated, but the common duct passed into a mass

of dense areolar tissue and enlarged glands in the portal fissure, through which its continuity could not be traced. The capsule of the liver was at many places adherent by firm fibrous bands.

The uterus was normal. The left ovary was as large as a walnut, rather soft, and nodulated. It contained a little semi-fluid dark blood; and its substance was soft and yellow, and exuded a creamy juice containing 'cancer-cells.' A cancerous nodule, the size of a pea, projected from the surface of the left ovary. The mesenteric and lumbar glands presented no abnormal appearance.

The lungs and heart were normal, with the exception of pulmonary congestion and patches of atheroma in the mitral flaps and in the commencement of the aorta. There were no cancerous deposits in either the spleen or kidneys.

CASE XLII.—*Cancer of Uterus and Liver—Ascites, but no Jaundice.*

On October 18, 1866, Charlotte D——, aged 56, was transferred to my care, having been for two months before under the care of the Obstetric Physician for cancer of the uterus. She was married, and the mother of nine children; the catamenia had ceased at the age of 49. Three years before she came under my care, she had an attack of what appeared to be gall-stones, sudden spasmodic pain in the right side, with vomiting and slight jaundice, and ever since she had suffered from a feeling of uneasiness and fulness below the right ribs. Twelve months before, she first noticed a slight but very offensive and persistent yellow discharge from the vagina, and ever since she had suffered from costiveness and pain in defæcation and some difficulty in micturition. On two occasions, nine months and three months before she came under my observation, she had rather copious uterine hæmorrhage, lasting for about a fortnight. Two months before, she first noticed her abdomen to swell, and she began to suffer from vomiting after food. She had been losing flesh for twelve, and rapidly for three, months.

On admission the patient was weak and emaciated, and her countenance was expressive of pain. There was extensive induration and ulceration of the cervix uteri and upper part of the vagina, with a fetid discharge. The abdomen was much distended, measuring $35\frac{3}{4}$ inches at the umbilicus, and exhibiting all the signs of fluid in the peritoneum. The liver was much enlarged in the

right mammary line, measuring $6\frac{1}{2}$ inches, and projecting fully 2 inches below the costal margin. The portion that could be felt was hard and tender, but had no feeling of nodulation. The superficial abdominal veins were slightly enlarged, but there was no jaundice. The tongue was moist and slightly furred, the vomiting had ceased, but the bowels had not acted for several days. The urine was loaded with lithates, but contained no albumen. There was no anasarca of the trunk or extremities. The pulse was 108 and feeble; there was no dyspnoea, and the cardiac and respiratory signs were normal, with the exception of slight dulness and fine crepitation at end of inspiration at base of right lung.

The patient was treated with bismuth and chloric ether, subcutaneous injections of morphia, mild laxatives, and a nutritious diet, with a small allowance of brandy. The vomiting did not return; but every night she suffered from intense pain in the abdomen, which was only partially relieved by the morphia injections. The belly slowly increased in size; the prostration became daily greater, until death occurred on October 30.

Autopsy.—The peritoneum contained several quarts of turbid serum, with flakes of soft lymph, chiefly on the fundus uteri and in the pouches before and behind. The cervix uteri was entirely destroyed by cancerous ulceration, which extended for $1\frac{1}{2}$ inch down the anterior wall of the vagina; the lower two-thirds of the uterus were infiltrated with cancerous matter. The lumbar glands were slightly enlarged from cancerous deposit, and in the portal fissure was a mass of enlarged cancerous glands pressing on the portal vein. The liver was of enormous size, weighing 115 ounces, and the portion opposed to the thoracic and abdominal wall measuring 7 inches. The entire liver was studded with numerous isolated nodules of cancer, from a pea up to a walnut in size, but none of them were much raised above the outer surface, so that the portion of the organ projecting beyond the ribs was perfectly smooth and even. On section, many of the nodules were found to be softening in the centre into a flaky serous fluid. On microscopic examination, the nodules, both at the circumference and in the centre, were seen to consist mainly of nuclear elements, with but few cells; the hepatic tissue intervening between the nodules was free from cancerous infiltration. The mucous membrane of the stomach and intestines was healthy, but small nodules of cancer, up to the size of a cherry, were scattered through the lower lobe of the right lung.

Although no opportunity was afforded for a post-mortem examination in the following case, the diagnosis, as I frequently pointed out in the wards, was sufficiently clear.

CASE XLIII.—*Cancer of the Liver, Lungs, and Cervical Glands—Jaundice and Ascites.*

John B—, aged 47, a cowman, was admitted under my care, on August 24, 1866. Twelve years before admission he had been confined to bed for a week with rheumatism; and two years before he had suffered for two months from severe pain at the epigastrium, usually worse after food. With these exceptions, he had enjoyed good health until eight weeks before he came to the hospital, when he was seized somewhat suddenly, while at work, with violent pain in the region of the liver and stomach, which had never ceased, although it had been sometimes more severe than at others. Eight days after this, he noticed that his motions had lost their colour, and that his urine was very dark, and after six more days, the conjunctivæ, and then the skin, became yellow.

On admission, the patient was weak and emaciated, and had intense jaundice of the entire surface. He complained of severe pain in the region of the liver, coming on in paroxysms, which would last for many hours, were sometimes attended by vomiting, and often prevented sleep. The liver was enlarged, measuring $5\frac{1}{2}$ inches in the right mammary line; in the epigastrium, it felt hard and obscurely nodulated, and was very tender. No tumour could be felt corresponding to the gall-bladder. There was neither ascites nor enlargement of the abdominal veins or spleen. The tongue was coated with a creamy fur; the bowels were costive; the motions clay-coloured and very offensive; the urine was of the colour of porter, and contained abundance of bile-pigment, but no albumen. The pulse was 96; the cardiac and respiratory signs were normal, and there was no dropsy.

The patient was treated with mineral acids and gentian, anodyne draughts with drachm doses of tincture of henbane, and mild laxatives.

On August 28, he first noticed a tumour in the left side of the

neck, immediately above the clavicle. It was about the size of a hen's egg, hard, nodulated, and slightly tender. This tumour increased in size, and soon became the seat of severe pain, like that in the liver. Patient also complained often of severe pain down the back, but there was no tenderness of the spine. Indian hemp and henbane failed to give relief to these pains, and on September 9, the subcutaneous injections of morphia were resorted to, at first with great benefit.

On September 5, ascites was first noticed which from this date continued to increase, and on September 24, both feet and the lower half of both legs were noted as swollen and œdematous. The tumour in the neck now filled up the whole of the lower triangle, and at its circumference were several large and movable glands quite distinct from the general mass: the patient vomited occasionally after his breakfast, and was becoming daily thinner and weaker.

On October 1, the patient was noted as vomiting almost every thing he swallowed. The pulse was 84, weak, and intermittent. The ascites and tumour of the neck continued to increase, the liver appeared larger and more distinctly nodulated, and the pains were only relieved by the morphia injections, which were repeated twice daily. There was no cough, and the respiration was slow and easy, but over the middle of the left lung posteriorly there was marked dulness over a space 3 or 4 inches square, with absence of vesicular murmur, but no friction or crepitation.

On October 5, the left arm and hand were noted as œdematous, and the vomited matter, which from the first had resembled yeast, was found to contain abundance of *sarcinæ*. A mixture was ordered every six hours, containing 10 minims of chloric ether, and 1 drachm of a saturated aqueous solution of carbolic acid in peppermint water.

The patient was now so weak that he obviously could not live many days, but his wife came, and insisted on removing him to the country.

The preparation which I now show you was removed from the body of a patient in this hospital while I was pathologist, and was exhibited to the Pathological Society (Trans. vol. xiii. p. 100). It

illustrates a rare mode of fatal termination of cancer of the liver.*

CASE XLIV.—*Primary Cancer of the Liver—Death from Hæmorrhage into the Peritoneum.*

Patrick S—, aged 50, became an out-patient at the Middlesex Hospital, under Dr. Greenhow, in August 1861. At a former period of his life, he had been very intemperate, and he had been in the habit of drinking a large quantity of spirits. For some months he had been losing flesh, and he had been suffering from occasional nausea and other dyspeptic symptoms, and from pains in the epigastrium. Dr. Greenhow discovered that the liver was enlarged and distinctly nodulated below the margin of the right ribs, and recognized the peculiar physiognomy characteristic of the cancerous cachexia. There was no jaundice, and little or no ascites; and nothing was observed to indicate an immediate fatal termination.

On August 26, the patient was brought to the hospital, and admitted under Dr. Goodfellow, his condition having become suddenly worse about two days before. His symptoms on admission were great prostration and cachectic countenance; marked jaundice of skin, conjunctivæ and urine; complete loss of appetite, urgent vomiting, intense pain and tenderness in the region of the liver, which was much enlarged, hard, and nodulated; abdomen much distended and fluctuating; small, rapid pulse.

No improvement took place; and on the day after admission, the patient vomited a large quantity of dark bloody-looking fluid.

During the night of the 27th, he fell into a state of collapse, which continued until death at 2.30 P.M. of the 28th of August.

Autopsy.—Moderate emaciation; marked jaundiced tint of conjunctivæ and skin and of the tissues generally, including the

* For additional cases, see Frerichs, *Dis. of Liver*, Syd. Soc. Trans. ii. p. 333; Murchison, *Path. Trans.* xiii. p. 102; also Budd, *Dis. of Liver*, 3rd ed. p. 396. In Frerichs's case, the hæmorrhage seemed to commence three days before death, and the appearances in the liver were very similar to those above described.

internal organs and the bones. Between five and six quarts of dark-red bloody serum were found in the peritoneal cavity, and lying on the upper surface of the right lobe of the liver towards its right extremity, between it and the diaphragm, was a dark coagulum of blood which weighed 5 ounces avoirdupois. The serous coat of the intestines, which was bathed by the bloody fluid, presented no abnormal injection or deposit of lymph. The liver weighed 72 ounces. The right lobe was relatively much enlarged, measuring 9 inches transversely, while the left lobe was much atrophied, and a mere appendage to the right, not exceeding $1\frac{1}{2}$ inch in its transverse diameter. The greater part of the diminutive left lobe was granular on the surface, and presented on section the appearances characteristic of cirrhosis. Corresponding to the lobus quadratus was a rounded mass, about the size of a large walnut, attached by a narrow pedicle, and likewise composed of cirrhotic glandular tissue. The whole surface of the right lobe of the liver was covered with prominent nodules, varying in size from a pea to a large cherry, the largest being elastic or presenting fluctuation. These nodules were most developed near the anterior margin of the right lobe on the upper surface. The coagulum on the surface of the right lobe was adherent at one spot near the right extremity of the organ, corresponding to one of the softened nodules, which was ruptured. The structure of the right lobe of the liver was extremely dense; and on making a section, it appeared to consist of two abnormal elements, a groundwork of firm grey scirrhus-looking tissue, infiltrated with a creamy yellowish juice, and containing a number of cavities up to the size of a cherry, filled with a soft pulpy bright yellow substance. The whole of the right lobe appeared to be made up of these abnormal elements, and scarcely presented at any part a trace of the natural glandular parenchyma or of bile-ducts. The scirrhus structure had encroached to some extent along the anterior margin of the left lobe.

On examining with the microscope the juice scraped from the denser scirrhus portions, it was found to contain a multitude of rounded, elliptical, and fusiform cells, up to $\frac{1}{800}$ of an inch in diameter, with one or sometimes two large nuclei about one-third the size of the cell; many of the cells, likewise, included brownish pigment granules. In the softened portions, similar cells were discovered, mixed up with a large quantity of oily and pigmentary matter, both inside and outside the cells.

The other abdominal organs were normal. The heart was

normal. The apices of both lungs were condensed and puckered, and contained encysted calcareous masses up to the size of a pea.

In Case XLV. the immediate cause of death was also probably hæmorrhage into the peritoneum. The preparation which I show you appears to be an illustration of that rare form of disease described by Dr. Bright and other writers as ‘fungus hæmatodes’ of the liver, where the growth projects greatly from the general surface of the organ. The transition between the secreting cells of the liver and the large cells of the growth determined by myself and Dr. Cayley, is likewise a matter of considerable pathological interest.

CASE XLV.—*Cancerous Tumour (Fungus Hæmatodes), projecting from upper surface of Liver—Hæmorrhage into Peritoneum.*

Luke T—, aged 57, was sent to the London Fever Hospital on January 20, 1868, supposed to be suffering from ‘fever.’ He had no friends, and could give no account of his previous history. On admission, he had a heavy stupid countenance, and his mind was confused. He was very emaciated; the pulse varied from 76 to 88, and was very weak. The tongue was dry and brown; the bowels were rather loose, and the abdomen was ascertained to be slightly distended, partly from tympanitis, but partly also from fluid in the peritoneum. The hepatic dulness appeared to be normal. There was an occasional cough, with thin frothy expectoration, and there was slight dulness over both lungs posteriorly and rather fine crepitation, but no tubular breathing. There were no night-sweats, and there was neither jaundice, dropsy, nor albumen in the urine.

The patient was treated with ammonia, and subsequently with iron and mineral acids, along with beef-tea, milk, and brandy; but the symptoms became gradually worse, the emaciation and ascites increased, there was frequent low muttering delirium; and on

February 2, slight jaundice was noted, but the motions still contained bile. The pulse rarely exceeded 80. The patient became every day weaker; but no fresh symptom of importance appeared. He died on February 16.

Autopsy.—The peritoneum contained between 3 and 4 quarts of dark sanguinolent fluid. The liver was separated from the diaphragm in front and from the anterior abdominal wall, for 2 or 3 inches, by a space filled with this sanguineous fluid. The suspensory ligament was elongated in a corresponding degree. The liver weighed 64 ounces; the capsule was slightly thickened and opaque, but the surface was smooth. On section it appeared unusually dense and tenacious. Projecting from the upper and back part of the right lobe was a rounded tumour, as big as a man's fist. This was embedded in a hollow in the diaphragm, to which it was so firmly adherent that part of it was left behind in removing the



Fig. 16 shows the microscopic appearances of the Tumour of the Liver in Case XLV. *a*. Large nucleated cells of various shapes, and some with a double nucleus; *b*, similar cells containing oil-globules; *c*, large cell containing bile-pigment; *d*, cells resembling in every respect the glandular epithelium of the liver; *e*, transitional forms between these last cells and the large cells.

liver. This tumour was of pulpy softness, and reflected over it was the thickened capsule of the liver, from the inner surface of which

the pulpy mass could be easily scraped with the handle of the knife. On section, there was seen to be a sharp line of separation between it and the dense tissue of the rest of the liver. The pulpy substance was torn with the greatest facility, and was very vascular, so that it was obviously the source of the blood in the peritoneum. On microscopic examination, it was found to be made up of large nucleated cells, with an average diameter of $\frac{1}{500}$ inch, or about three times that of an hepatic gland-cell. The cells were rounded, pyriform, or caudate, and each contained one or sometimes two nuclei, with much fine granular matter; some were full of oil-globules, and some contained brown pigment-granules exactly like what is seen in the gland-cells of the liver. Along with these large cells, which were much the more numerous, were others of smaller size, and not to be distinguished from the secreting cells found in other parts of the liver (fig. 16).

The stomach and intestines were healthy; the walls of the heart were thin and soft; both lungs were firmly adherent, and much congested in their dependent parts. There was nothing noteworthy in any other organ.

Case XLVI. is an illustration of cancer implicating the liver, without producing any symptoms or signs which could lead to its existence being suspected during life. One of the supra-renal capsules also was destroyed by cancer, and yet there was no vomiting or bronzing of the skin. It is now well known that the supra-renal capsules may be destroyed by cancer, without any of the symptoms of Addison's disease resulting, so that these symptoms must be ascribed, not to the destruction of the capsules, but to the morbid process by which this is effected.

CASE XLVI.—*Cancer of Vertebrae, Supra-renal Capsule, Liver, and Lung—No Symptoms of Disease of Liver.*

Alfred T—, aged 55, was admitted into the Middlesex

Hospital, under my care, on January 28, 1868. He was very weak and emaciated, and not very connected in his replies. Seventeen years before admission, he contracted syphilis, followed by constitutional symptoms, but his 'present attack' commenced only three months before admission, with severe pain in the spine, accompanied by emaciation and weakness.

His symptoms while under observation were as follows:—Progressive emaciation and debility, and anæmic chlorotic colour of face; but no jaundice, or bronzing of skin, or discoloration of mucous membrane of mouth, or perspirations. Persistent pain and tenderness on pressure over spinous process of the third and fourth lumbar vertebræ, but no sign of tumour or of paraplegia, excepting retention of urine for the last two or three weeks of life. Tongue dry, red, and fissured; no vomiting; constipation alternating with diarrhoea. Abdomen distended and tympanitic, with slight tenderness on deep pressure to the left of the umbilicus: a few days before death the abdominal swelling subsided, and the aorta could be felt passing down along the spine, but there was no appreciable tumour. The hepatic dulness was 4 inches in the right mammary line: at no time was there tenderness, or a feeling of nodulation in the region of the liver, or ascites. The pulse varied from 84 to 120, and was always small and weak; the cardiac dulness was diminished; there was at no time any cough or expectoration, and at the time of admission, no notable sign of mischief could be discovered in the lungs. The urine was alkaline and contained phosphates, but no albumen or bile-pigment. The temperature was either normal, or but slightly increased. Throughout the mind was confused, and there was a tendency to low muttering delirium, increasing towards death, which occurred on March 22.

Autopsy.—There was a soft cancerous tumour of the bodies of the third and fourth lumbar vertebræ, projecting about half an inch from the surface, chiefly on the left side, where it invaded the texture of the psoas muscle, and encroaching about half-way to the spinal canal, which, as well as the spinous processes, appeared normal. There was cancerous enlargement of the lumbar and bronchial glands, and a mass of soft cancer, the size of a large walnut, compressing a large branch of the pulmonary artery in the upper part of the lower lobe of right lung. The liver was not enlarged, and its lower margin did not project beyond the edge of the ribs, but it contained from a dozen to twenty isolated cancerous nodules

from the size of a pea to that of a walnut, several of which were excavated in the centre. One of these nodules was in a portion of liver which was firmly adherent to the right supra-renal capsule. The latter organ was greatly enlarged, and converted into a mass of hard cancer, measuring $2\frac{1}{2}$ inches in diameter. The left capsule, the kidneys, and the brain presented nothing abnormal.

The following case came under my notice while I was House Surgeon in the Edinburgh Royal Infirmary. It is an example of a rare form of cancer implicating the liver, but causing no symptoms of hepatic disease.

CASE XLVII.—*Melanotic Cancer of the Penis, Lymphatic Glands, Liver, Pleura, etc.*

James L—, aged 54, a butler, was admitted into the Royal Infirmary, Edinburgh, on February 4, 1851. He was a tall, robust man; his hair was dark brown, and his eyeballs were remarkable for their prominence, and for a bluish tint of the sclerotics. Attached to the lower and outer surface of the prepuce, and extending a considerable way along its free margin, there was a tumour, the size of a chestnut, of a dark brown, almost black colour, and with its surface nodulated and covered with a fetid, dirty yellow, puriform discharge. When pricked with a pin, it bled profusely, and it was often the seat of acute pain, especially during, and for a short time after, micturition. It had been growing for two years, and commenced as a small black wart on the outer surface of the prepuce, about an inch from its free margin: this wart for six months remained stationary, but afterwards increased more rapidly. On reflecting the prepuce, which was done with some difficulty, there were displayed on the surface of the glans several warty excrescences of a bluish black colour, and varying in size from that of a pin's head to that of half a pea. In each groin, there was a swelling of the size of a hen's egg, which had first appeared about three months before admission.

For three months the patient had complained of dyspnœa and cough; and on examining the chest, the left side presented a uniform bulging, measuring fully 1 inch more in circumference than the

right. There was also on this side marked dulness on percussion, imperfect expansion, and absence of the natural respiratory murmur and of vocal thrill. The apex of the heart was displaced to the left margin of the sternum. The physical signs of the right lung were normal. Pulse 90, and very feeble.

After this, the patient got rapidly worse; he lost all relish for food, and became very prostrate. The fits of dyspnœa increased in frequency and in severity, lasting sometimes for several hours, and dulness with suppression of the respiratory murmur was observed over the base of the right lung. The tumour on the penis and the swellings in the groins increased slightly in size. There was no jaundice, ascites, or enlargement or pain of the liver.

On the morning of March 26, he had an unusually severe attack of dyspnœa; the pulse was 84, and almost imperceptible; the extremities cold; face livid and the eyeballs more prominent. These symptoms continued until death on the evening of the 27th.

Autopsy.—The tumour on the penis presented on section a smooth, black surface, yielding on section a copious inky juice. The lumbar, inguinal, and femoral glands, were enlarged and infiltrated with black matter; and some of them were entirely converted into a pulpy black fluid. The lymphatics of the spermatic chord contained 1 or 2 small melanotic nodules. Along the whole of the abdominal aorta, there was a chain of enlarged glands. Some of these exhibited, on section, a black pulpy mass, while others, which were but slightly enlarged, presented the normal glandular structure, with circumscribed brownish black points. The hypogastric and sacral lymphatics were normal.

Left pleura distended with several quarts of fluid tinged with blood and black pigmentary matter, which pushed the apex of heart towards right side. Scattered over the whole of the parietal and pulmonary pleura, were masses of a dark deposit, varying in size from the smallest appreciable point to half an inch in diameter, and, for the most part, presenting a circular outline. The largest of these nodules projected about one-sixth of an inch from the surface of the pleura; the smallest were not appreciably elevated, presenting a punctiform appearance not unlike the shading of a chalk drawing. The large nodules were almost black, while the punctiform deposit had a brownish black tint, tinged more or less with purple. Most of the nodules were covered by the epithelial layer of the pleura, but at the back part of the cavity, where they were confluent and aggregated into flattened masses, this mem-

branous lining was at some places wanting, and the masses exhibited a pulpy irregular surface, and yielded on pressure a large quantity of dark juice very like liquid sepia. The left lung was completely compressed and carnified. At the reflection of the pleura from the root of the lung upon the ribs, there was a layer of recently extravasated blood, at some parts half an inch in thickness. Right pleura contained a few ounces of fluid similar to that in left, and its surface exhibited nodules of deposit of the same character, but much less extensive. Embedded in the substance of the right lung, were a few circumscribed black nodules, the largest about the size of a cherry: around them, the pulmonary tissue was normal and crepitant. The bronchial glands were all black, but not much enlarged. In the posterior mediastinum, the glands were greatly enlarged, and a cluster of them, forming a mass the size of an orange, was situated in the angle of bifurcation of the trachea, in front of the œsophagus. The deep cervical glands contained black pigment.

Between the mucous and muscular coats of the œsophagus there were one or two rounded nodules, the size of a barley-corn, containing black pigment; the rest of the alimentary canal and the mesenteric glands were normal. On the surface of the liver there were seen about a dozen nodules of the black deposit, about one-third of an inch in diameter; and numerous similar masses were found embedded in the substance of the organ which was but slightly increased in size. In the spleen there was a single mass of black deposit, the size of a pea.

The kidneys contained in various parts of their cortical substance melanotic nodules, the size of a swan-shot. Between the muscular and mucous coats of the bladder and of the urethra were a few black nodules the size of barley-corns.

Chemical Examination of Melanotic Matter.—The following analysis of the pigmentary matter was made by Dr. James Drummond:—

‘It was insoluble in water, alcohol, and ether. When treated with hydrochloric, nitric, and sulphuric acids, it was dissolved; the solution being nearly colourless. When chlorine gas was passed through it suspended in water, it was bleached to a certain extent, but not entirely. When boiled with potash, it dissolved, with disengagement of ammonia. The ultimate analysis yielded the following result:—

Carbon	67·01
Hydrogen	6·45
Nitrogen	11·45
Oxygen	8·36
Ash	6·73
	<hr/>
	100·00

‘The ash consisted, in great part, of peroxide of iron.’

Microscopic Examination of the Melanotic Deposit.—The dark juice from the tumour on the penis was found to contain a large quantity of granular matter of a sienna brown colour. The granules were solid and angular, and refracted the light strongly. Acetic acid produced no change upon them; but strong nitric acid rendered them much lighter. Mixed up with these granules were a few nucleated cells, having a circular or oval outline, and a diameter of about $\frac{1}{500}$ of an inch. Some of the cells were more elongated, and one or two exhibited a caudate appearance. Most of them were loaded with the coloured granules, which quite obscured all appearance of a nucleus. In some of the cells, however, which contained little or none of the coloured granules, one and sometimes two nuclei could be detected with one or two distinct nucleoli. When a small particle of the tumour was torn out with needles and examined, it exhibited a network of fine filamentous tissue, infiltrated through the meshes of which were the elements of the dark coloured juice just described.

The melanotic deposits in the pleura, lumbar and inguinal glands were also subjected to careful microscopic examination, and were all found to possess a structure similar to that of the tumour on the penis.

The morbid conditions considered in this and the preceding lectures comprise almost all the enlargements of the liver ordinarily met with in practice; but there are others of rarer occurrence, with the clinical characters of which we are yet imperfectly acquainted.

1. *Tubercular Enlargement.*—I have already detailed to you the particulars of a case where a con-

siderable enlargement of the liver was found to be mainly due to tubercular deposit (Case XXX, page 140). The case was one of acute tuberculosis, and possibly the enlargement of the liver which occurs in this condition may occasionally be due to tubercle, rather than to fatty or waxy deposit. There are no means, however, by which at present tubercular enlargement can be distinguished during life, and its discovery would probably in no way modify the prognosis or the treatment.

2. *Lymphatic Enlargement*.—In leukæmia and in certain other affections where there is a general tendency to enlargement of the lymphatic system, the liver may be found studded with greyish white new formations, not unlike miliary tubercles,* but occasionally as large as peas, which may cause the whole organ to be enlarged. Structurally these formations resemble minute lymphatic glands and they are believed to be developments in connection with the lymphatic system. When therefore in a case of leukæmia the liver is found to be enlarged, the enlargement may be due either to this cause or to simple hypertrophy (see page 53); but it will call for no special treatment apart from that of the general condition.

3. *Multilocular Hydatid Tumour*.—This is a very rare form of tumour, composed of numerous small hydatid vesicles embedded in an alveolar structure of

* Frerichs, Dis. of Liver, Syd. Soc. Transl. ii. p. 222.

fibrous tissue, and not like an ordinary hydatid enveloped in a parent cyst. Its clinical characters have not yet been sufficiently studied, but are very different from those of an ordinary hydatid tumour of the liver. The tumour is not smooth, fluctuating and painless, but is nodulated, hard and tender. In most cases also there is enlargement of the spleen with jaundice and signs of fluid in the peritoneum, in consequence of the tendency which the disease has to implicate the bile-ducts and the portal vein. In most cases the tumour ultimately suppurates in the centre and induces symptoms of hectic fever. The disease for which it would be most readily mistaken is a cancerous tumour, and like cancer it sometimes runs a rapid course of a few months; but in other cases the tumour has been known to exist for ten years and more prior to death. The treatment recommended for ordinary hydatid tumours is obviously inapplicable here, and our efforts must be limited to the relief of symptoms as they arise.

4. *Enlargements of the Gall-bladder.*—The gall-bladder may be enlarged from various causes, so as to form a tumour attached to the liver, and appreciable through the abdominal parietes. The causes, the symptoms, and the treatment of these enlargements we shall reserve for consideration in a future lecture (Lect. XII.).

Lastly, the liver has been noticed to be enlarged and tender in cases of a rare and remarkable skin eruption known as *Vitiligoidea*. The enlargement is

usually accompanied with jaundice, and both may last for years. Of the cause of the enlargement we are as yet ignorant, but we shall have occasion to recur to the subject when we come to speak of Jaundice (Lect. VIII.).

LECTURE VII.

CONTRACTIONS OF THE LIVER.

SIMPLE ATROPHY—ACUTE OR YELLOW ATROPHY—CHRONIC ATROPHY—
(CIRRHOSIS—SIMPLE INDURATION—RED ATROPHY.)

GENTLEMEN,—In previous lectures I have described to you the normal limits of the area of hepatic dulness (p. 3), as well as the principal causes of apparent and real enlargement of the liver, with the means of recognizing them. We have now to consider the chief causes of a diminution in the area of hepatic dulness, and their distinctive characters. And in the first place you must remember, that the area of hepatic dulness often appears diminished, although the organ in reality retains its normal weight and bulk.

SPURIOUS CONTRACTIONS OF THE LIVER.

The main conditions likely to induce an apparent diminution in the size of the liver are as follows:—

1. Tympanitic distention of the bowels, and particularly of the transverse colon and stomach, may prevent the lower margin of the liver being felt, and diminish the area of hepatic dulness in several ways.

a. A portion of stomach or intestine distended

with gas may become interposed between the surface of the liver and the abdominal parietes.

b. When the lower margin of the liver is thin, and when there is excessive tympanitic distention of the subjacent bowels pushing the liver forwards and rendering the abdominal parietes tense, the lower edge of the liver may escape detection on palpation, and its dulness or percussion may be imperceptible.

c. In excessive tympanitis the antero-posterior diameter of the abdominal cavity is increased, and the lower portion of the liver may be elevated so that a smaller portion of it than is natural is in contact with the abdominal parietes.

In one or more of these ways the normal hepatic dulness may be diminished, or may even entirely disappear, so that the pulmonary sound is immediately succeeded by that of the bowel. The liver may thus appear greatly diminished, although its size may in reality not be altered. You will find a remarkable case of this sort recorded by Dr. Bright, where, on opening the body, neither the liver nor the colon presented itself to view, but, in their stead, the convolutions of the small intestines, which were found to have come completely in front of the liver, the colon and the omentum doubling over the liver and pressing it back, so as to have made deep furrows on its anterior surface.* The fact of hepatic contraction being of this spurious character ought always to be suspected under the following circumstances:—

* Abdom. Tumours, Syd. Soc. Ed. p. 259.

a. The very fact of there being tympanitic distention of the bowels ought to suggest caution in inferring the existence of real atrophy of the liver from a diminished area of hepatic dulness. The same caution is necessary in cases of ascites. The fluid in the peritoneum may push up the bowels which may be only moderately distended with gas, but which may thus come to produce the same result as more extensive tympanitis; and this fallacy is of the greater importance inasmuch as ascites is a common consequence of real atrophy of the liver.

b. Variations in the extent of hepatic dulness at different times is a character of spurious atrophy of the liver most useful in diagnosis. The dulness of the liver will vary in its extent according to the amount of gas in the stomach and bowels. The diagnosis will therefore be facilitated by oft-repeated examinations, and particularly by examinations made before meals, and after the bowels have been cleared out by a purgative.

c. Variations in the extent of hepatic dulness at different places is not uncommon in cases of spurious atrophy. Tympanitic distention of the stomach and bowels may diminish or obliterate the hepatic dulness in the median and right mammary lines, but is not likely to affect it materially in the axillary or dorsal lines. Occasionally too the space where the hepatic dulness is obscured may be even more circumscribed, as when a knuckle of intestine intervenes between the liver and the abdominal wall.

d. There is an absence of other signs or symptoms of real disease of the liver, but the possibility of there being ascites independently of hepatic disease, already referred to, must be kept in view.

2. General or partial *accumulations of gas in the peritoneal cavity*, such as may result from perforation of the stomach or bowels, may obscure, to a greater or less extent, the area of hepatic dulness; but usually the nature of these cases will be sufficiently clear from—

a. The arched tympanitic distention of the abdominal parietes; and

b. Antecedent history of peritonitis from perforation.

3. The hepatic tissue may be *preternaturally soft*, so that the organ may fold on itself and collapse against the spine and the back part of the abdomen, and be covered more or less in front by the stomach and bowels which may not be abnormally distended with gas. I have already pointed out to you that in fatty degeneration, the enlargement may from this cause appear to be increased, a larger portion than natural of the liver being in apposition with the abdominal wall (page 44); but if the folding be carried a stage further so as to permit the super-position of bowel, a contrary result may take place. Lastly, in acute atrophy of the liver, the organ is not only reduced in size, but it may be so soft as to collapse against the spine, all trace of it disappearing from the abdominal wall in front.

Keeping in view these sources of fallacy, which are, on the whole, more calculated to mislead than the sources of fallacy in the case of enlargement (see page 9), we may proceed to consider the causes of real atrophy of the liver, which may be conveniently arranged under the three following heads:—

I. Simple Atrophy.

II. Acute or Yellow Atrophy.

III. Chronic Atrophy; under which head will be included the diseases commonly designated ‘Cirrhosis,’ ‘Simple Induration,’ and ‘Red Atrophy,’

I shall now endeavour to describe to you the leading clinical characters and the appropriate treatment of these several forms of atrophy.

I. SIMPLE ATROPHY.

By ‘simple atrophy,’ is understood a diminution in the size of the liver, independent of any alteration in its structure, except a diminished size of the lobules, which may be so small as to be distinguished with difficulty, the cut surface presenting a smooth appearance and often a uniform tint. The liver in this state may be reduced to one half of its normal weight and bulk, or even less. Although this condition of liver is not of much practical importance, more or less of it is far from uncommon, and ignorance of its nature and characters may lead to errors in diagnosis. You will recognize this form of atrophy then by the following characters:—

1. The circumstances under which it occurs. These are mainly two, viz. Old Age and Inanition.

a. Old Age—Simple atrophy, has been sometimes described as ‘senile atrophy.’ With the advance of life, the tendency of the various organs and tissues throughout the body is either to degenerate or to waste. In some persons, the several forms of *degeneration* (fatty, calcareous, &c.), predominate; while in others we observe a simple *wasting*. In the latter case, the power which prevailed over the waste of the body in childhood and youth, and which maintained the balance in the vigour of manhood, has failed, and waste now prevails over development. In most cases you will find that the reduction of the liver in old age is in advance of that of the body generally, and occasionally the liver is reduced by senile atrophy to one half of its normal size and weight.

b. Inanition may also induce simple atrophy. There is little or no supply to compensate for the constant waste. When you remember the increase in the bulk of the liver produced by every meal (see page 120), you will readily understand how, in cases of inanition, the liver often wastes out of proportion to the rest of the body. It is difficult to say why it is that the effect of wasting diseases is in some persons to cause wasting of the liver, while in others it leads to the accumulation in the organ of a large quantity of oil (see p. 49). Inanition may arise in two ways, either from an insufficient supply of food, or, as certainly, from diseases which interfere with

the assimilation of food. Accordingly you will find simple atrophy of the liver extremely common in the bodies of persons who have died of stricture of the pylorus, or of stricture of the œsophagus or cardiac orifice of the stomach. I shall relate to you immediately the particulars of a patient, aged 54, with a cancerous tumour of the lower end of the œsophagus, in whom the area of hepatic dulness was reduced to one half of the normal standard, and whose liver after death was found to weigh only 32 oz., instead of 54 oz., the average weight for his age (Case XLVIII). You will remember also the case of Samuel H., aged 63, who died of a cancer of the œsophagus involving the apex of the left lung, and whose liver was very small and weighed only 42 oz.; and the case of Eliza P., aged 48, who died of cancer of the pharynx, and whose liver weighed only 35 oz. All these were good examples of simple atrophy.

c. External Pressure by tight lacing, pleuritic or pericardial effusions, circumscribed peritoneal exudations, or enlargement of those portions of the bowel nearest to the liver, may likewise produce simple atrophy of the liver. The atrophy, however, under these circumstances is usually partial and is of little clinical importance, unless the bile-ducts or large blood-vessels have been subjected to the pressure.

2. There is an absence of any sign of hepatic disease or derangement. With the diminution in the size of the liver, there is, no doubt, a loss of functional power, but sufficient secreting tissue remains for the

work to be done. Care, however, must be taken not to mistake for symptoms of diseased liver, those of the primary disease on which the atrophy depends.

Simple atrophy of the liver requires no special *treatment* beyond that adapted to the circumstances under which it occurs.

The following case will serve to impress on your memories the clinical characters and post-mortem appearances of simple atrophy of the liver. The case is also interesting as an illustration of cancerous and tubercular deposit taking place simultaneously, of which other examples have been reported by Mr. Sibley,* Dr. Bristowe,† and myself.‡ It is difficult to account for these cases on the ordinarily accepted view, that tubercle and cancer depend on a ‘peculiar diathesis,’ regulating the nature of the exudation, for then the diathesis must vary in different parts of the same body.

CASE XLVIII.—*Coexistence of Cancerous Stricture of the Œsophagus, with recent Tubercle in the Lungs. Simple Atrophy of the Liver.*

Augustus T—, aged 54, a tailor, was admitted into the Middlesex Hospital, under my care, on July 24, 1863. He was of average height, and naturally of spare habit. He had led a very intemperate life, drinking large quantities of gin, but he had always enjoyed good health, until about four weeks before admission, when he began to suffer from sickness, coming on immediately after eating, sometimes even before he thought the food had been swallowed. He had never observed blood in the vomited matters, but he had rapidly lost both flesh and strength.

* Med. Chir. Trans. vol. xlii. p. 149.

† Trans. Path. Soc. vol. x. p. 284.

‡ *Ibid.* vol. xv. p. 104.

On admission he was very emaciated, and had an anxious expression of countenance. He could swallow solid food; but it was usually rejected, either immediately or within a few minutes. He also brought up from time to time large quantities of clear acid fluid. He complained of pain between the shoulders, but there was no tenderness of the spine, and no abnormal physical sign in either lung. The abdomen was nowhere tender, and nothing like a tumour could be felt at any part of it. The hepatic dulness was much diminished, not exceeding $2\frac{1}{2}$ inches in the right mammary line. The splenic dulness was normal, and there was no ascites or jaundice. The tongue was furred and the bowels costive. The pulse was 61 and feeble; there was no abnormal bruit with the heart, and no anasarca, nor albumen in the urine.

All remedial measures failed to relieve the vomiting, and the patient got rapidly thinner and weaker, while the hepatic dulness was reduced to 2 inches. On August 30, the vomiting abated, but this was due to the patient's taking scarcely any nourishment. He died on September 7. At no period of his illness did he suffer from cough.

Autopsy.—Entire absence of fat beneath the integuments and throughout the body. The œsophagus, $1\frac{1}{2}$ inch above the cardia, had its calibre narrowed to that of a goose-quill for about half-an-inch. A hard tumour, the size of half a walnut, was firmly attached to the constricted portion, and formed part of its posterior wall. The mucous membrane corresponding to this presented a puckered cicatrix-like appearance. The substance of the tumour was dense, fibrous, white, and slightly translucent, and dotted over with softer, more opaque, yellow specks. It yielded an opaque juice on scraping. On microscopic examination, the firmer portions of the tumour were found to contain numerous 'cancer cells,' varying in size up to $\frac{1}{450}$ inch in diameter. They were rounded, elliptical, and caudate, and contained one or two large nuclei with a diameter about one-third of that of the cell. Some of the cells had smaller cells in their interior. In the softer portions of the tumours, the cells were ill-defined and mixed with much oily and granular matter. Neither the bronchial glands, nor the lymphatics in the neighbourhood of the tumour were enlarged. The stomach was small, but otherwise normal. The liver presented the ordinary characters of simple atrophy; it weighed only 32 ounces;

its outer surface was smooth; the only abnormal appearance seen on section was that the acini were reduced to one-half of their usual size. The secreting cells were small, and contained scarcely any oil, but were otherwise normal. The spleen weighed only 3 ounces; the kidneys were also small and anæmic, but in other respects normal.

Both lungs were very small, the right weighing $9\frac{3}{4}$ ounces, and the left $8\frac{3}{4}$ ounces. The apices of both were firmly adherent to the thoracic walls, and marked externally with cicatrices. Several cretified deposits as large as peas, as well as one or two small cavities with thick walls and containing pus, were disclosed on cutting into the cicatrices. Scattered through the upper lobes of both lungs were a number of translucent greyish granules, isolated and collected into groups, as large as a hazel-nut, and presenting all the naked-eye and microscopic characters of miliary tubercles. The heart weighed only $6\frac{3}{4}$ ounces, and was destitute of fat, but in other respects was normal.

II. ACUTE OR YELLOW ATROPHY.

This is a rare but very remarkable disease, in which the liver becomes rapidly atrophied with the development of jaundice and cerebral symptoms, and where after death, what remains of the organ is found to be extremely soft and yellow, with no appearance of lobules, and with the secreting cells in a great measure, or wholly, broken up into granular matter and oil-globules. The rarity of the disease in this city is attested by the fact that although a brown tongue and delirium constitute a certain passport for the transmission of all diseases to the London Fever Hospital, out of about 15,000 cases admitted during the last six years, the only example of the disease which has been noticed is the one of which I shall narrate to you the particulars immediately. The disease,

however, is one of the most interesting that can engage your attention, and may be recognized by the following clinical characters :—

1. Premonitory symptoms are noticed in many cases, but they are variable in their nature and sometimes absent. The most common are those of gastro-enteric catarrh, such as furred tongue, nausea, and loss of appetite, occasional vomiting and irregular bowels—diarrhoea or constipation, with slight pyrexia. At other times, the patient complains only of rheumatic pains, of an uneasy sensation in the region of the heart, or of a feeling of uneasiness which he is unable to define. These symptoms may last three or four days, or as many weeks, but withal there is not, as a rule, thought to be much amiss, while in not a few cases the patient has no feeling of indisposition until the supervention of symptoms of a more decided character.

2. Jaundice is invariably present, and is usually the first symptom that attracts attention to the patient's condition. The jaundice, however, is rarely intense, and is sometimes confined to the upper part of the body. Like the jaundice of pyæmia (see p. 148), it appears to be due to a poisoned condition of the blood, and is independent of any obstruction of the bile-duct, and consequently bile is still found in the stools.

3. A rapid diminution in the area of hepatic dulness is one of the most remarkable features of the disease. In the course of a week or ten days, one-third, or even

more than one-half of the liver may disappear (see fig. 17). Bright has recorded a case where the liver after death weighed only nineteen ounces, and its weight in the case which I shall bring under your notice was only twenty-eight ounces. It has been lately suggested that the atrophy in these cases is after all a chronic process, though unattended by symptoms

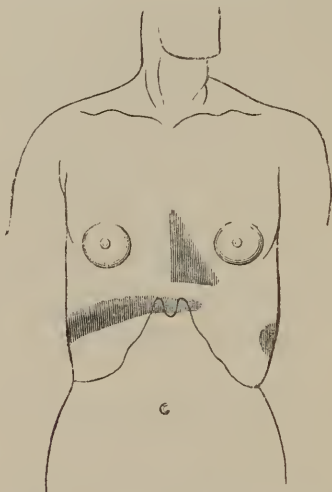


Fig. 17. Area of Hepatic Dulness in Mary Ann M—— (Case XLIX.), on the day before death.

until the final explosion ; but this view is opposed by most of the known facts in reference to the etiology of the disease, and also by the circumstance that the atrophy may be traced by percussion during life. Careful examination of the gland after death shows that the atrophy is due to a destructive process commencing at the circumference of the lobules and

advancing to the centre, as the result of which the secreting cells disappear and in their place we find nothing but granular matter and oil. The disease, in fact, appears to be nothing more nor less than an acute fatty degeneration, resulting from a diffuse inflammatory process; for, previous to bursting, the cells may often be seen distended with oily and granular contents. During life the atrophy of the liver may appear greater than it really is, because the gland is not only reduced in size, but also softened, so that it folds upon itself and collapses towards the vertebral column, the space corresponding to it in front being occupied by intestines containing gas.

4. Pain at the epigastrium and in the region of the liver is present in most cases. This pain often comes on spontaneously, and can almost always be elicited by pressure, even when the patient is almost unconscious. There is rarely, however, any tympanitic distention of the abdomen.

5. Vomiting occurs in most cases, the vomited matters consisting of the ingesta with mucus or bile, but often also containing blood, and resembling the 'black vomit' of yellow fever. The bowels are described as being usually constipated, but in the case from which this liver was taken (Case XLIX.) there was a considerable amount of diarrhœa. The stools not unfrequently contain blood and are very offensive.

6. The area of splenic dulness is usually increased except in cases where the portal system has been

drained by diarrhœa or by hæmorrhage from the stomach or bowels.

7. The cerebral symptoms of the 'typhoid state' constitute one of the most frequent and striking peculiarities of acute atrophy. As a rule, they appear simultaneously with the jaundice, but occasionally not for two or three weeks subsequently. At first there is headache, with despondency, irritability, and great restlessness; and this condition is succeeded by low muttering delirium, tremors, subsultus, muscular rigidity, and carphology, retention or incontinence of urine, involuntary passage of fæces, stupor, coma, and convulsions. These symptoms are independent of any lesion of the brain or of its membranes; but, like the analogous symptoms in typhus fever and in the typhoid state generally, to which I have directed your attention on a former occasion,* they result from the circulation through the brain of blood poisoned by the accumulation in it of urea and other products of metamorphosis which ought to be eliminated by the kidneys.

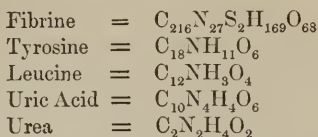
8. Acute atrophy of the liver is not accompanied by much febrile excitement. The pulse varies. In cases ushered in with gastro-enteric catarrh the pulse is usually accelerated at first, but falls to the normal standard, or below this, on the appearance of jaundice, but again rises on the supervention of cerebral symptoms. In the case which I shall relate

* On the Pathology and Treatment of the Typhoid State in different Diseases. Abstract of Lecture in Brit. Med. Journ. Jan. 4, 1868.

to you it rose to as high as 144. Occasionally it has been found to be very variable at different periods of the day. Careful observations on the temperature in acute atrophy are still wanting. Most writers, such as Bright and Alison, have noted that the skin is cool. According to Frerichs, the temperature is increased at the onset of the disease and again during the period of nervous excitement, and in Case XLIX., where there was peritonitis, it rose to as high as 101° F. After the appearance of cerebral symptoms, the tongue is almost invariably dry and brown, and the teeth crusted with sordes, exactly as in a bad case of typhus fever.

9. The urine undergoes important changes. Its quantity is not materially altered; it is of acid reaction; and its specific gravity varies from 1012 to 1024. Its colour is usually dark, but the ordinary reaction of bile-pigment may be faint or indistinct. It often contains a small quantity of albumen or even blood; but after the removal of the urinary pigment, it yields no reaction of bile-acids to Pettenkofer's test. The most remarkable alterations, however, consist in the great diminution or even total disappearance of the urea and uric acid, and also of the chlorides, sulphates, and earthy phosphates, and the substitution of two new substances of a peculiar nature, leucine and tyrosine. These substances are products of the metamorphosis of matter intermediate between the proteine principles (albumen and fibrine) at one extreme, and the less complex bodies, urea, uric acid,

kreatine, &c. at the other, as will be seen by a comparison of the following formulæ:—



Leucine and tyrosine, in the crystalline forms represented in the annexed figures (figs. 18, 19, and

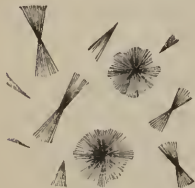


Fig. 18. Microscopic needle-shaped crystals of tyrosine adhering in bundles and in stellate groups.

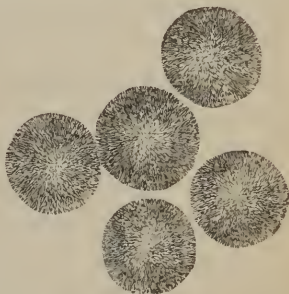


Fig. 19. Microscopic globular masses composed of acicular crystals of tyrosine.

20), are found in the tissues of the liver, spleen, and kidneys in cases of acute atrophy, and they are usually also secreted in large quantity in the urine from which they separate as a distinct deposit on standing, or they may be obtained by evaporating a few drops of the urine on a glass slide. The detection of these crystalline bodies in the urine of a case of jaundice may be said to clinch the diagnosis of acute atrophy of the liver, but the failure to detect them

must not exclude acute atrophy from the diagnosis. For instance, they were not present in the urine of Case XLIX., at all events in such quantity as to reveal their existence by simply evaporating the urine, although they were found in considerable quantity in the liver and kidneys after death. It is true that in this case death was accelerated by acute peritonitis; had the patient survived a little longer, leucine and tyrosine would probably have been found in the urine.

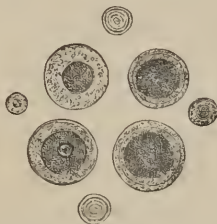


Fig. 20. Microscopic laminated crystalline masses of leucine.

10. Hæmorrhages are very common, and particularly hæmorrhage from the stomach or bowels. Blood is often vomited in large quantity. Petechiæ and vibices may appear on the skin, or in rarer cases there is uterine hæmorrhage or epistaxis.

11. Pregnant females, who constitute a large proportion of the cases, almost invariably abort or miscarry before dying.

12. The circumstances under which acute atrophy of the liver occurs constitutes not the least interesting part of its clinical history. The causes of the disease still require investigation, but I shall briefly

mention those that are at present known. Among *predisposing* causes, then we have —

a. Age. Most persons attacked with the disease are under the middle age. Of 31 cases collected by Frerichs, 26 were under 30, and all but 2 under 40.

b. Sex. The disease is much more common in females than in males. Of the 31 cases collected by Frerichs, 22 were females.

c. Pregnancy must be regarded as a predisposing cause, for of the 22 female patients referred to by Frerichs, one half were attacked while pregnant. From the third to the sixth month is the most common period of pregnancy at which the disease shows itself. In the pregnant state it is said to be frequently associated with fatty degeneration of the kidneys and albuminous urine.

d. Dissipation, including drunkenness and venereal excesses, have been known to precede the disease in a considerable number of cases.

e. Constitutional syphilis appears to be a predisposing cause in some cases. Most writers on syphilis have noted the frequent occurrence of jaundice about the commencement of what is known as the secondary stage; and in some (probably a small proportion) of these cases the jaundice is due to acute atrophy.*

Among causes that appear to act more directly in *exciting* the disease are the following:—

a. Nervous influences, such as severe mental emo-

* See Lebert in Virchow's Archiv. 1854, 1855; Andrew in Path. Trans. xvii. p. 158; and Fagge, *Ib.* xviii. p. 138.

tions, and particularly fear and grief. Sir Thomas Watson, in his lectures, states that scores of instances are on record, where jaundice has suddenly appeared under such circumstances, and adds that these cases are often fatal, with head symptoms, convulsions, delirium, or coma, supervening upon the jaundice.* In these cases an impression made upon the nervous system appears to be directed to the liver and to derange its nutrition.

b. Malaria. There are other cases where the disease has apparently resulted from some malarious poison, acting probably through the blood and the nervous system. Instances have been recorded by Graves,† Budd,‡ and others, where several cases of what appears to have been unquestionably this disease occurred in the same house; and when it is considered what a rare disease acute atrophy is, it is impossible to escape from the view that in these cases there must have been some local cause to which all the patients were subjected in common.

c. The blood-poisons of typhus fever and allied diseases have been known to give rise to acute atrophy of the liver.§ Jaundice is a very rare complication of typhus and scarlet fever, but in more than one instance where it has occurred,|| I have found crystals of leucine and tyrosine in the tissue of the

* Lectures on the Practice of Physic, 3rd ed., ii. p. 557.

† Clin. Lect. 2nd ed. ii. p. 255.

‡ Op. cit. 3rd ed. pp. 255, 270.

§ See Frerichs' Treatise on Diseases of Liver, Syd. Soc. Ed. i. p. 235

|| Treatise on the Continued Fevers of Great Britain, 1862, p. 194.

liver and kidney. The liver in these cases has been in a state of fatty degeneration, but without marked atrophy. Most writers on the yellow fever of the tropics have described fatty degeneration of the liver as one of its most characteristic lesions; but observations are still wanting as to the presence or absence of leucine or tyrosine in the urine and in the tissues of the kidneys and liver.

d. Lastly, it seems not improbable that in some cases of acute atrophy, the cause may be, as suggested by Dr. Budd, some special poison engendered in the body itself by faulty digestion or assimilation.* The nervous influences already referred to may possibly contribute to the development of such a poison.

Treatment.—In acute atrophy of the liver, all treatment has hitherto proved unsatisfactory. The disease, after the supervention of cerebral symptoms, is in most cases fatal, although well authenticated instances are on record where patients have recovered after falling into a state bordering on coma. It may be well, therefore, to enumerate those remedial measures which have appeared most useful, or which seem indicated by our knowledge of the pathology of the disease.

1. Purgatives. In several instances which have been reported as occurring in Ireland, patients, in the same house with others who have died, have recovered after active purging in conjunction with leeches and blisters to the head.† Dr. Budd also states that in several

* *Op. cit.* p. 265.

† See cases by Dr. W. Griffin, of Limerick, in *Dub. Journ. of Med. and Chem. Science*, 1834, and by Dr. Hanlon, in *Graves, loc. cit.*

cases of jaundice which he believed to be of this nature, he has found advantage from a combination of sulphate of magnesia (3j), carbonate of magnesia (gr. xv), and spiritus ammoniæ aromaticus (3ss) given three times a day.

2. After the supervention of cerebral symptoms all measures calculated to promote the elimination of urea and uric acid from the system deserve a trial. It is in this way perhaps that purgatives have proved beneficial, and that warm baths, hot air baths, diaphoretics, diuretics, and colchicum may also be expected to do good.

3. In cases where there is extensive hæmorrhage from the stomach or from other mucous membranes, ice or astringents may be necessary.

4. It is in the early stages, however, of the malady, before the occurrence of cerebral symptoms, that most advantage may be expected from treatment. Cheering society, holding out hopes of recovery, change of scene, anodynes to procure sound sleep, attention to the condition of the stomach and bowels, and the nitro-muriatic acid, with the infusion of gentian, or some other vegetable bitter, are the measures which appear best calculated for averting those terrible cerebral symptoms from which so few recover.

The liver, which I show you here, was taken from the body of a patient who recently died under my care, in the London Fever Hospital, and who presented the symptoms of acute atrophy of the liver in a

typical form, excepting that no leucine or tyrosine was found in the urine passed the day before death. These substances, however, were detected after death in the tissue of the liver and kidneys, and the former of these organs presented all the anatomical characters peculiar to the disease. It may be worth mentioning, however, that both Dr. Cayley and myself failed to find either leucine or tyrosine in the fresh liver and kidneys, although they were present in large quantity after these organs had been immersed for some days in spirit.

CASE XLIX.—*Acute Atrophy of the Liver—Acute Peritonitis—Leucine and Tyrosine in the Liver and Kidneys, but none detected in the Urine.*

Mary Ann M——, a sempstress, aged 19, was admitted into the London Fever Hospital, on the evening of February 13, 1868, and was seen by me on the following morning. She was unmarried. Her father was a German, but she had been born and brought up in London. Her sister was not aware that she had suffered from any mental trouble, and believed that her catamenia had been regular; there was no history of syphilis. There had been no other case of illness in the house from which she came. She had been quite well until the middle of January, when she began to complain of loss of appetite and nausea, and, after ten days, her skin was noticed to be slightly yellow. A week before admission she took to her bed, complaining of pain in her stomach, aggravated by any movement, but unattended by vomiting. For about a fortnight before admission her bowels had been relaxed three or four times a day, the motions at first being yellow, but latterly green. Three days before admission she began to be 'light-headed.'

On the morning after admission the following note was taken:—'Patient is a well-nourished girl, and has deep jaundice of the skin and conjunctivæ. She is scarcely conscious, and can give no account of herself. Since admission she has been very restless and

delirious, often screaming out loudly. The pupils are much dilated, but equal. There is no eruption on the skin, which feels dry and hot, the temperature in the axilla being 101° F. The pulse is 116 and weak. The cardiac and respiratory signs are normal. The tongue is dry and brown, and since admission there has been frequent vomiting of a dark brownish fluid evidently containing blood. The bowels have acted several times, and from the nurse's account, who describes the motions as having been very dark, watery, and offensive, they probably also contained blood. The abdomen moderately distended, and tympanitic; pressure upon it does not seem to cause pain, but the respiration is thoracic, and there is an obscure thrill, as from fluid, on tapping both flanks. The hepatic dulness is greatly diminished, not exceeding $1\frac{1}{4}$ inch in the right mammary line, and its lower margin being fully 2 inches above that of the ribs (see fig. 17, p. 226). The urine has been passed in bed, but the bladder is now full.'

About 2 pints of urine were drawn off by catheter, which had the following characters. It was acid, and had a specific gravity of 1015. It had a dark greenish-brown colour, but presented the reaction of bile-pigment in only a faint degree. Heat produced no change on it; but, on adding nitric acid, after boiling, it became turbid, as well as very dark. Nitrate of urea could be obtained from it in only very small quantity, but no crystals of leucine or tyrosine could be detected, either as a separate deposit on standing, or after evaporation of a few drops of the urine in a watch-glass. Unfortunately the urine was thrown away before it could be submitted to a more careful analysis.

The patient was ordered a mixture containing nitric acid, nitric ether, and nitrate of potash, with milk, beef-tea, and four ounces of gin.

She nevertheless became rapidly worse, although she was less noisy and delirious, and seemed to sleep a good deal at intervals. The diarrhoea continued, the motions being passed in bed, and being still liquid and very offensive, but of a light yellow colour. In the evening of the 14th the pulse was 144; respirations 132 and thoracic; temperature in axilla 100.8° . She continued much in the same state, and died, without any convulsions, at 7.50 a.m. on the following morning, five days after the first appearance of cerebral symptoms.

Autopsy.—Body well nourished. Much purple lividity of inte-

guments, and deep jaundiced hue of skin and of every tissue of the body. No scars on genitals or in groins.

Three or four pints of slightly turbid serum in peritoneum. Considerable fine vascular injection of the serous covering of the small intestines, and particularly of that of the duodenum. The peritoneum of the intestines and of the liver was also coated at many places with a thin film of recent lymph, easily separated. The stomach and intestines were distended with gas, and the liver was completely hidden below the right ribs, not more than an inch of it being opposed to the thoracic wall. The organ was extremely small; its largest diameter measuring $6\frac{1}{2}$ inches, and the antero-post. diameter of the right lobe only 5 inches. It weighed only 28 ounces, or exactly one-half of the standard weight for the girl's age. It was very flabby, and the outer surface was wrinkled, but free from any granular or nodular irregularities. The substance of the gland was extremely friable, and of almost pulpy consistence, and presented, at some places, a tolerably uniform rhubarb-yellow colour, with scarcely any appearance of lobules, and at other parts a similar yellow colour, interspersed with red. Under the microscope, there was found a large quantity of free oily and granular matter, with globular masses of leucine, and bundles of needles of tyrosine, and also, more especially at what corresponded to the centres of the lobules, entire secreting cells of large size, and loaded with oil-globules and dark greenish-yellow pigment. The bile-ducts were patent throughout; they were not dilated, and their lining membrane presented no tinge of bile, although the gall-bladder contained about a teaspoonful of dark-green viscid bile, which could be squeezed out through the cystic duct. The contents of the intestine consisted throughout of a very pale yellowish pulp. The mucous membrane of the bowels was nowhere ulcerated. The spleen was of normal size, but rather soft. Both kidneys were slightly enlarged, extremely soft, and tinged with bile-pigment. The renal epithelium contained a large quantity of fine granular matter; crystals of both leucine and tyrosine were detected in the renal tissue. The bladder was empty and the uterus was unimpregnated. There was much hypostatic congestion of both lungs. The pericardium contained more than an ounce of yellow serum; the heart was healthy; the blood was dark and fluid. Excepting an increased amount of serosity in the lateral ventricles and beneath the arachnoid, neither the brain nor its membranes presented anything abnormal.

III. CHRONIC ATROPHY.

Under this head it will be convenient to consider several diseases, which, in their etiology and anatomical characters, are essentially distinct, but which often present symptoms so similar that it may be impossible during life to distinguish them. The diseases I refer to are these :—

I. Cirrhosis, or the so-called ‘gin-drinker’s liver,’ in which the liver becomes reduced in size in consequence of an atrophy or slow destruction of the secreting tissue, but where the fibrous tissue is increased in amount, so that the organ is preternaturally dense and firm. The outer surface also presents a granular or nodulated character, which has earned for the disease the designation of ‘hob-nailed liver,’ and on section the organ presents firm fibrous bands, including the remains of vessels and bile-ducts and surrounding islets of yellow* secreting tissue. The capsule also is sometimes thickened and adherent to surrounding parts. This disease can almost invariably be traced to the abuse of strong spirits, and especially to the habit of drinking them neat, and accordingly it is most common in those countries and towns where such a habit prevails. (Case L.)

* This yellow colour is due to the large quantity of yellow pigment contained in the secreting cells. It is from this character that the term cirrhosis (*κίρρós*, yellow), is derived, and the application of the term to diseases of other organs, such as the lungs, or kidneys, which resemble cirrhosis of the liver, not in the yellow colour, but in the fibroid condensation of the tissue, is obviously inappropriate.

II. Hyperæmia, from obstructed circulation in cardiac and pulmonary diseases, causes, in the first place, enlargement of the liver (see p. 123) ; but when of long standing, the enlargement is followed by an opposite condition of atrophy. The liver also becomes firm, tenacious, and finely granular, and presents an appearance which has frequently been mistaken for cirrhosis. The depressions however correspond to the centre of the lobules, whereas in true cirrhosis, they are at the circumference. The atrophy is due to the pressure exerted by the distended capillaries of the hepatic vein on the surrounding secreting cells. These cells disappear, so that the central portions of the lobules sink down, while the portions occupied by the branches of the portal vein project as fine granulations. After a time the atrophy extends to the circumference of the large branches of the hepatic vein, so as to cause extensive depressions, and new connective tissue is developed around the vessels, imparting to the organ a greater degree of firmness, and more or less obstructing or obliterating the minute branches of the portal vein. This condition of liver is not uncommon in cases of valvular disease of the heart of long standing.

III. An atrophy of the liver where the organ also presents a granular or nodulated outer surface resembling what is seen in true cirrhosis, but where the fibrous tissue is not increased, so that the liver instead of being preternaturally dense, is softer than in health. In some, if not all, of these cases there is no history of spirit-drinking. (See Case LIV.)

IV. An atrophy of the liver, resulting from frequent attacks of peri-hepatitis, or inflammation of the capsule. In these cases the capsule becomes greatly thickened and is often connected to surrounding parts by firm bands of adhesion. Fibrous bands also pass from the thickened capsule into the interior of the liver, which on section often presents a dense, smooth, uniform surface, with the outline of the lobules more or less obliterated. This condition of liver has been described by some writers under the name of 'simple induration,' and is not uncommon in patients who have suffered from constitutional syphilis; it is also met with occasionally in cases of valvular disease of the heart of long standing, and in connection with inflammation of the right pleura, ulceration of the mucous membrane of the stomach, or various diseases of the secreting tissue of the liver itself. In these cases inflammation is propagated to the capsule of the liver through the diaphragm, along the coronary ligament, or from the subjacent glandular tissue. When the disease has a syphilitic origin, the surface of the contracted liver is often marked by cicatrix-like depressions or deep fissures, and fibroid tumours are found in the interior; under other circumstances, the outer surface is smooth, and it never presents the hob-nailed character of true cirrhosis. Now and then the fibroid tissue developed in the portal fissure from the products of inflammation produces constrictions of the bile-duct or portal vein.

V. In the next place there is the 'chronic

atrophy' of Frerichs, or the 'red atrophy' of Rokitansky. Here there is no nodulation or granulation of the outer surface, and not necessarily any thickening or adhesions of the capsule, but the secreting tissue contains a large quantity of blood, and presents on section a dark-brown or bluish-red colour, a rather firm consistence, and a homogeneous appearance with little or no indication of a division into lobules. The secreting cells are often smaller than natural, and loaded with brown pigment-granules. The atrophy of the organ is general, although its thickness often preponderates over the other dimensions, and occasionally there is a broad rim of atrophied hepatic tissue round the edge. The entire organ has been known to weigh only 24 ounces. But the most important anatomical character is the destruction of the ramifications of the portal vein, the branches of which terminate in blind club-shaped extremities, so that the organ cannot be minutely injected from the portal vein. This form of atrophy is occasionally seen in connection with simple and cancerous ulcerations of the stomach and intestines, or, with a deposit of black pigment in the minute vessels of the liver, in the bodies of persons who have suffered long or often from intermittent or remittent fevers.

In all of these diseases there is one anatomical character in common, viz.—a destruction to a greater or less extent of the minute branches of the portal vein in the interior of the liver. To this cause must be attributed the clinical symptoms in which during

life they so closely resemble one another. The prominent symptoms in all of them are those of obstructed portal circulation. It will be convenient therefore to describe to you in the first place the typical symptoms in a case of true cirrhosis, and afterwards to mention those circumstances which may serve to distinguish from it the other forms of chronic atrophy.

I. First then let us consider the clinical characters of true cirrhosis. This is a very chronic disease; its history usually extends over several years, and may be conveniently divided into two stages, that which precedes, and that which follows, the destruction of the minute branches of the portal vein.

A. *First Stage*.—The disease at its outset is usually insidious, and the symptoms throughout the first stage are often obscure. Except there be complications, there is no fever, and the chief symptoms are as follows:—

1. Symptoms of disordered digestion are the most common, such as loss of appetite, furred tongue, flatulence and pain after food, languor and depression of spirits, irregular action of the bowels, and in some cases a tendency to vomit the food.

2. A dull pain with slight tenderness in the right hypochondrium is usually present.

3. In many cases also there is slight enlargement of the liver. This was doubted by the late Dr. Todd, but the observations of Bright,* Budd,†

* Guy's Hosp. Rep. 1st Ser. vol. i. p. 612.

† Dis. of Liver, 3rd Ed. p. 151.

and Frerichs,* seem to leave little doubt on the matter.

Although these symptoms may not be sufficient for the diagnosis of cirrhosis, yet when they are present in a person who is known to have been addicted to spirit-drinking, the existence of that disease in its early stage ought always to be suspected.

4. Now and then the disease sets in with symptoms of a more striking character. Sometimes it commences with vomiting and purging, and other symptoms of gastro-enteritis. In other cases there are all the symptoms of acute congestion of the liver—fever, with much pain and tenderness in the right hypochondrium, unmistakeable enlargement of the organ, nausea and vomiting, jaundice, and high-coloured urine containing bile-pigment and depositing lithates.

B. *Second Stage*.—The symptoms in this stage are usually well marked, and are mainly due to the obstructed portal circulation.

1. The area of hepatic dulness is diminished (see fig. 21). It may be reduced to one-half of the normal standard, or even less. The atrophy is usually greatest in the left lobe, all dulness from which may entirely disappear. The dulness also of the right lobe may be reduced to a greater extent than would be accounted for by the actual decrease of the liver, its lower edge being tilted up by the pressure of fluid in the peritoneum, or gas in the bowels, which at the

* Dis. of Liver, Syd. Soc. Ed. ii. pp. 35, 37.

same time increases the antero-posterior diameter of the abdomen, and diminishes the extent of liver which is in apposition with the abdominal parietes. It is well to remember also that in rare cases of cirrhosis, even at an advanced stage, the hepatic dulness is increased, owing to the coexistence of waxy or amyloid deposit. (See pp. 25, 193.)

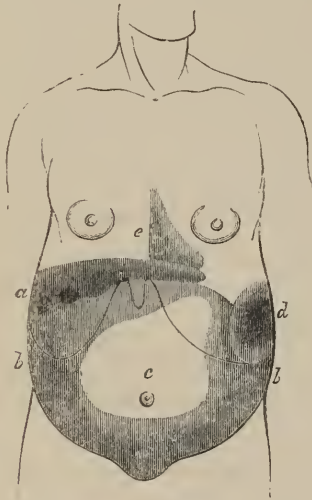


Fig. 21. Shows the Hepatic and Ascitic Dulness in Cirrhosis. Thomas B——, Case L. (p. 260).

a. Dulness of contracted liver. *b.* Fluid in peritoneum causing bulging of flanks.
c. Tympanitic bowels. *d.* Enlarged spleen. *e.* Heart.

2. The nodulated or hob-nailed character of the outer surface of the liver may sometimes be felt through the abdominal parietes, and lend assistance to the diagnosis. More commonly, by the time that the irregularity of the surface is sufficient

for this purpose, the organ is so small that its lower margin is concealed by the ribs. In those cases where there is a co-existence of cirrhosis and waxy disease, and where in consequence the liver is enlarged, its outer surface may be marked by large nodules, separated by deep fissures. This nodulated character may be easily distinguished through the abdominal parietes, and, as I have already shown you (page 193), may be readily mistaken for cancer. In regard to diagnosis also, it is necessary to keep in view the possibility of a nodulated character of the liver being congenital, or the result of obliteration of large branches of the portal vein. In the latter case, the surface of the liver presents deep fissures, caused by the atrophy of the glandular tissue, which had formerly been supplied with blood by the obliterated vessel.

3. Ascites. A dropsical collection of fluid in the peritoneum, without any pain or tenderness of the abdomen, is one of the most common results of portal obstruction, and is met with oftener in cirrhosis than in any other disease of the liver. The fluid in the peritoneum is a clear yellow serum, containing a large quantity of albumen, but without blood or inflammatory products. In consequence of the distention of the veins that return the blood from the peritoneum, the serous portion of the blood transudes through the walls of the vessels into the peritoneal cavity. When once it appears, it persists and gradually increases. When the amount of fluid

is large, it may compress the inferior vena cava, and the iliac veins, and thus produce secondary œdema of the legs: but it is a peculiarity of dropsy from uncomplicated portal obstruction, that the ascites precedes and preponderates over dropsy elsewhere. A great amount of ascites may also interfere with the action of the diaphragm, and cause much embarrassment of the breathing, but it is distinguished from the ascites produced by cardiac disease, by the fact that the dyspnœa follows and never precedes the ascites. Again you must remember that the pressure exerted on the renal veins by a large quantity of fluid in the peritoneum may lead to the appearance of albumen in the urine independently of any disease of the kidneys, the albumen disappearing on the removal of the pressure after the operation of paracentesis.

Frerichs noted ascites in only twenty-four out of thirty-six cases of cirrhosis: patients with cirrhosis may no doubt die in various ways before there is any ascites, but in the advanced stages of the disease ascites is rarely absent.

4. Enlargement of the spleen causing an increased area of splenic dulness is another common consequence of the mechanical obstruction to the circulation through the liver, although on the whole, it is less common than might at first be imagined. It is present in about one-half of the cases. Its absence is sometimes attributable to fibrous thickening or calcification of the capsule, interfering with the dilatation of its contained vessels, and at other

times to an excessive drain from the gastro-intestinal mucous membrane, caused by diarrhœa or hæmorrhage.

5. Enlargement of the superficial veins of the abdomen, especially on the right side and between the sternum and the umbilicus, is another result of the impediment to the current of portal blood, usually observed in advanced cirrhosis. It must be borne in mind, however, that a similar enlargement may be noticed in extreme ascites from any cause, owing to the pressure of the fluid on the vena cava inferior. In this case, however, there is usually also varix of the veins of the lower extremities.

6. Hæmorrhoids not uncommonly result from the same cause as the enlargement of the abdominal veins, and often precede the other signs of portal obstruction.

7. Hæmorrhages from the mucous membrane of the stomach and bowels occasionally occur, and are sometimes profuse and fatal. The minute vessels of the mucous membrane rupture, owing to their extreme distention with blood, and to the collateral circulation not being sufficiently developed. Epistaxis, purpuric spots on the skin, ecchymoses about the punctures of the lancet in cupping, and other hæmorrhages which are obviously independent of a mechanical cause and are probably the result of some altered condition of the blood, are not uncommon in the advanced stages of cirrhosis.

8. Gastritis and enteritis also frequently occur in

the course of cirrhosis, the congested mucous membrane being excited to inflammation by causes which would otherwise be inert. Their occurrence is marked by abdominal tenderness, pain and vomiting after food, and obstinate diarrhœa, with more or less fever. It is in such cases that we often find after death the typical anatomical characters of catarrhal inflammation, with hæmorrhagic erosions in the mucous membrane of the stomach, appearances which by the inexperienced are not unfrequently believed to result from some irritant poison. In those cases where waxy degeneration co-exists with cirrhosis, there is often profuse and obstinate diarrhœa from waxy disease of the bowels.

9. Pain in the region of the liver is not a prominent symptom of true cirrhosis. In the early stage there is sometimes a dull heavy pain, with some tenderness in the right hypochondrium arising from congestion, and throughout the disease there may be acute pain and tenderness of a temporary nature, resulting from intercurrent attacks of peri-hepatitis, but in the intervals of these attacks, there is but little pain or tension in the region of the liver.

10. Decided jaundice is a rare symptom in cirrhosis. In the early stage, there may be jaundice from congestion, but when jaundice shows itself subsequently, it is usually the result of some complication, such as catarrh of the ducts, or enlarged glands in the fissure of the liver compressing the bile-duct. Occasionally also, deep jaundice shows

itself shortly before the fatal termination, and is accompanied by purpura spots, a dry brown tongue, delirium, coma, convulsions, and other symptoms of blood-poisoning.

But although decided jaundice is rarely met with in cirrhosis, there are few patients who do not, in the advanced stage of the disease, present a persistent sallowness of the complexion, with a darker areola round the eyes, although the motions are coloured by bile, and the urine contains little or no bile-pigment. Care must be taken not to confound with this sallowness the bronzed appearance of the face from exposure to the sun in hot climates, or the waxen complexion of anæmia.

11. The digestive functions are sometimes in a tolerably normal condition, but more commonly there is loss of appetite, with flatulence and constipation, or the symptoms of gastro-enteritis already referred to.

12. In all cases the advance of the disease is marked by progressive emaciation and debility, and in most cases the patient dies by exhaustion, the intellectual faculties remaining clear to the last. At other times death is due to pneumonia, œdema of the lungs, or acute peritonitis, or is preceded by the symptoms of general blood-poisoning already mentioned.

13. In most cases the diagnosis of true cirrhosis will be aided by being able to trace a previous history of spirit-drinking, and especially a habit of drinking brandy, rum, gin, or whiskey, in an undiluted form. It is not quite certain, indeed, that true cirrhosis ever

results from any other cause, for, although it is said to have been met with in young children, and even in some of the lower animals, such as cattle and pigs, it is very possible that the disease in these cases has been one of the other forms of chronic atrophy to which I have called your attention.

The clinical characters of the other forms of chronic atrophy are very similar to those of true cirrhosis as above described. I may now mention to you, however, the circumstances which during life may often enable you to distinguish them, although practically their distinction is a matter of no great importance as regards either prognosis or treatment.

II. The contraction which results from mechanical obstruction of the circulation differs from true cirrhosis in the following respects :—

1. There are the previous history and the existing physical signs of serious disease of the heart or lungs.

2. Dyspnœa precedes the ascites.

3. Although the ascites will preponderate over the signs of dropsy elsewhere, it is preceded by œdema of the legs, which persists.

4. The contraction of the liver is usually preceded by more marked symptoms of congestion—enlargement, pain, and jaundice—than those observed in the early stage of most cases of cirrhosis ; and unless there be coexistent peri-hepatitis, the contraction is rarely so great as in true cirrhosis.

5. The existence of true cirrhosis will be rendered still more improbable if there be an absence of any history of spirit-drinking.

III. The atrophy of the liver in which the organ presents a nodulated outer surface like that of true cirrhosis, but which has no increase of the fibrous tissue, and in consequence has its tissue either of natural consistence or softened instead of being preternaturally dense, cannot be distinguished from true cirrhosis by any clinical characters with which I am acquainted. In several cases, however, where I have met with this form of the disease, there has been no history of spirit-drinking (see Case LIV.), so that possibly this negative character may be of some use in diagnosis.

IV. In what is called simple induration of the liver, the main points of difference from true cirrhosis are as follows :—

1. When the edge of the liver can be felt, it is usually smooth and hard, instead of being nodulated. An exception, however, must be made with regard to those cases which have a syphilitic origin, and where as already stated the surface of the liver may be marked by large nodules separated by deep fissures.

2. Pain and tenderness in the region of the liver are greater and more constant than in true cirrhosis.

3. The circumstances under which simple induration is known to occur are important in the diagnosis. Thus there is :—

a. A previous history of general peritonitis, of

ulceration of the stomach, of inflammation of the right pleura, or of constitutional syphilis.

b. An absence of any history of spirit-drinking.

c. Occasionally simple induration is met with in cases of valvular disease of the heart, in conjunction with the second form of chronic atrophy already referred to.

V. Clinically it will always be difficult, and oft-times impossible, to distinguish 'red atrophy' (see page 241) from cirrhosis or simple induration. As in these affections the disease runs a chronic course, there is a great diminution in the area of hepatic dulness, and there are the symptoms of portal obstruction, viz. ascites, enlarged spleen, &c. The surface of the liver, when it can be felt, differs from that of cirrhosis in being smooth, but the chief indication of its existence are the circumstances which precede the symptoms of portal obstruction.

a. There is no history of spirit-drinking.

b. There is no dyspnœa or valvular disease of the heart; but

c. In many cases there is an antecedent history of ague or malarious remittent fever.

d. In others there has been a history of dysenteric or other ulceration of the intestinal canal.

Treatment of Cirrhosis.

A. *In the early stage.*—From the difficulty of its recognition, or from the patient not applying early for medical advice, an opportunity is rarely afforded of

treating the disease at its commencement; but if from the presence of the phenomena already cited, there is reason to suspect the existence of incipient cirrhosis, the indications for treatment will be as follows:—

1. Attention to the diet is the object of first importance. This ought to be spare, and to consist of such articles as milk, eggs, farinaceous substances, and plainly cooked fish or meat. Alcohol in every form, all hot spices, and everything that is rich or indigestible ought to be strictly interdicted. Too often, in spite of all advice, the patient persists in his ruinous habits.

2. Regular exercise in the open air ought always to be enjoined.

3. The bowels ought to be kept freely acting by occasional doses of saline purgatives, such as the sulphate or citrate of magnesia, the bitartrate of potash, or Cheltenham or Püllna water. The action of these remedies may also be assisted by small doses of calomel, blue pill, podophyllin or rhubarb. (See page 126.)

4. Alkalies or their salts with the vegetable acids are also often useful. Small doses of the bicarbonate, citrate, or tartrate of potash or soda, or of the artificial Vichy salt, may be given in solution two or three times a day; or the patient may have recourse to the mineral waters of Vichy, Ems, Vals, or Carlsbad.

5. If there be much pain or uneasiness in the

region of the liver, it will be necessary to employ dry cupping, or mustard and linseed poultices over the right hypochondrium. If the pain be great, it will be most speedily relieved by a few leeches applied to the right hypochondrium, or round the anus. Patients, however, addicted to spirits cannot bear much depletion, which is apt to induce delirium tremens.

6. In cases where all pain and uneasiness have subsided, but where the liver remains enlarged, the various preparations of iodine—the iodide of potassium, the compound tincture of iodine, or the syrup of the iodide of iron, or the rubbing of iodine ointment into the side—and also the bromide of potassium—are sometimes of use. If they fail in reducing the liver, the mineral acids, and bitter tonics, and the nitro-muriatic acid bath (see page 128) ought to receive a trial.

B. In the *second stage* of the malady there is no treatment which can restore the portion of liver that has been lost, or remove the obstructions to the portal circulation. All that can be done is to counteract the effects of the disease, and support the patient's strength; but in effecting the latter object you must beware of aggravating the primary cause of mischief.

1. The diet ought to be more nutritious than that which is permissible in the early stage, but still non-stimulant in its character. Hot spices, and everything that is indigestible, must still be avoided. Alcoholic stimulants, in every form, ought only to be allowed with great caution. As a rule, the patient is better without

any; but in the advanced stages, when there is great ascites and the heart's action is weak, small quantities may be advisable. Some patients also will insist on having a daily allowance of stimulant. Under these circumstances, dry sherry diluted with water, and the light wines which contain least alcohol, such as claret and hock, are the best.

2. Tonics, such as nitro-muriatic acid, taraxacum, gentian, quinine, nux vomica, strychnia, and iron, ought to be given from time to time, to improve the appetite, digestion, and general strength.

3. Purgatives will be necessary when the bowels are sluggish. The compound colocynth and henbane pill, with the addition of a small quantity of blue pill, or podophyllin, or some of the saline purgatives which are suitable in the early stage, may be given when required. Sulphur will be the most appropriate purgative when there are hæmorrhoids.

4. For the ascites, you must have recourse to diuretics and purgatives. As diuretics you may give the acetate or bitartrate of potash, with nitric ether and decoction of broom-tops, and this mixture may be advantageously combined with a pill containing squill (Pulv. Scillæ, gr. jss), digitalis (Pulv. dig. gr. ss) and blue pill (gr. ij), to be taken twice or three times daily. This pill is one that has enjoyed a long and merited reputation for the treatment of dropsy in this hospital. Diuresis will also sometimes be induced by fomenting the abdomen with an infusion of digitalis of about four times the usual

strength. The muriate and benzoate of ammonia, in doses of from ten to twenty grains, are also sometimes useful as diuretics, and may be advantageously combined with taraxacum. By acting on the kidneys there is good reason for believing that we can diminish or retard the increase of the ascites; but when the ascites is already great, it must be confessed that diuretics are of little avail either in reducing it or in preventing a still further increase. Purgatives are then of more use, and those ought to be selected which have most power in increasing the watery exhalation from the mucous membrane of the bowels, such as salines, the compound jalap powder, gamboge, and elaterium. An excellent purgative is an electuary composed of compound jalap powder and confection of senna. These purgatives ought always to be given in the morning before food, so as not to sweep away the food which has been digested but not assimilated. Drastic purgatives, however, must be given with some caution, for obstinate enteritis is one of the natural results of the disease, and sometimes causes death by exhaustion.

But notwithstanding the use of diuretics and purgatives, the ascites, as a rule, slowly increases, and sooner or later the belly attains such a size, as to seriously embarrass the breathing. Then, and not till then, you must draw off the fluid by the operation of paracentesis. The tapping may have to be frequently repeated, but the rule is always to delay it as long as possible, until in fact there is danger of the respiratory function

becoming seriously interfered with by the pressure of the fluid. The fluid usually collects again rapidly, and the frequent repetition of the operation will only increase the patient's exhaustion, in consequence of the great drain of albumen from the blood which the reaccumulation entails. By paracentesis the pressure on the renal veins is removed, and thus after the operation, not only does albumen often disappear from the urine, but diuretics will act more readily, and may retard the reaccumulation of fluid in the peritoneum. When there is much œdema of the legs, as well as ascites, both may be relieved by acupuncture of the legs, or by making an incision through the skin into the areolar tissue, about an inch above the inner ankle of each leg, followed by poultices, according to the plan originally recommended by Dr. Mead. The quantity of serum which will sometimes drain away from these punctures or incisions, to the great relief of the patient, is surprising.

5. Intercurrent attacks of peri-hepatitis may require local depletion, cataplasms and opium.

6. Attacks of gastritis will demand sinapisms and blisters to the epigastrium, with ice and lime water, or bismuth and hydrocyanic acid internally; the diet should be restricted to milk and farinaceous articles; and when the vomiting is urgent, it should consist of small quantities of milk alone. In enteritis it may be necessary to apply a few leeches round the anus, and to administer the vegetable or mineral astringents with opium, and in particular the acetate of lead with morphia. In slight cases,

however, it is inexpedient to check the purging too hastily or completely.

7. When copious hæmorrhage occurs from the stomach or bowels, the remedies indicated are ice, astringents, and especially tannic acid, opium, and the application of leeches round the anus.

8. Flatulence is often a source of so great distress, and aggravates so much the dyspnœa arising from the ascites, as to call for treatment. It will often be relieved by the various ethers and the essential oils of peppermint, anise, or cajeput, by vegetable charcoal, or by galbanum and assafoetida. Inasmuch, however, as it is probably due to decomposition from deficient or deteriorated bile, those remedies will be found most useful which act by checking decomposition, such as creosote, turpentine, or carbolic acid. From ten to thirty minims of carbolic acid, with an equal quantity of spirit of chloroform, may be given in an ounce of peppermint water, or a pill containing one drop of creosote.

9. When cerebral symptoms and other signs of blood-poisoning supervene, no treatment will probably be of any avail, but recourse may be had to purgatives and blisters to the scalp.

10. The principles of treatment which have been recommended for cirrhosis are applicable to the other forms of chronic atrophy of the liver, with the following modifications.

a. In atrophy of the liver resulting from disease of the heart, the treatment of the symptoms of obstructed

portal circulation must be subsidiary to that of the more important primary disease in the chest. Even here, however, although alcoholic stimulants will be more necessary than in true cirrhosis, they must be given with caution (see page 126).

b. In cases with a marked syphilitic history, and where there is reason to infer the presence of syphilitic peri-hepatitis, mercury and iodide of potassium may be expected to be of service.

I shall now narrate to you a few cases of chronic atrophy of the liver, in illustration of the remarks which I have made respecting its pathology and treatment. The first is a good example of true cirrhosis from spirit-drinking.

CASE L.—*History of Spirit-drinking and Symptoms of portal Obstruction—Dense fibrous granular Liver—True Cirrhosis.*

Thomas B—, aged 52, a butcher, was admitted into the Middlesex Hospital, under my care, on April 30th, 1867. His father and mother had lived to an advanced age; a brother and sister had died of consumption. He was a large, stout man, had always enjoyed good health until about two years ago, when he began to suffer from flatulence, and during the last year he had also complained of shortness of breath, disturbed sleep, chilliness, and occasional palpitation; at the same time he had noticed some swelling of his legs and abdomen. He thought that this swelling had gone away after two or three weeks, but about four months ago it had reappeared, and had since increased considerably. He said that it had first reappeared in the left leg, but possibly it was not greater in this situation than elsewhere, and his attention had been more directed to it, owing to a vesication which appeared over the left ankle. He had never been troubled with hæmorrhoids, but

on several occasions during the last two years, he had vomited about a teacupful of black blood. His habits had been always intemperate; he had drunk freely both spirits and beer.

On admission, the patient exhibited an emaciated, sallow countenance, with a slightly jaundiced tint of the conjunctivæ. There was great œdema of the lower extremities and scrotum, and evidence of a large accumulation of fluid in the peritoneum, the umbilicus being quite obliterated, and the girth of the abdomen at this part measuring 46 inches. There was also considerable enlargement of the subcutaneous veins of the abdomen, especially on the right side. The hepatic dulness was diminished, in the right mammary line being less than 3 inches (see fig. 21, p. 245); the splenic dulness was increased, measuring vertically 4 inches. There was no jaundice, except the slight icteroid tint of the conjunctivæ above referred to. There was no tenderness of the abdomen, nor vomiting; the tongue was moist with a white fur; the bowels were very costive, often not acting for a week; the heart's action was feeble, but the dulness and sounds were normal. Pulse, 120. Crepitus, at some places rather fine, was audible over the lower half of both lungs, back and front, but there was no decided dulness, nor tubular breathing; respirations, 36. The urine was acid, it contained no albumen, but yielded a decided reaction of bile-pigment: specific gravity, 1032.

The patient was treated with purgatives and diuretics, but no improvement took place in his condition; on the contrary the dropsy increased in the legs, and on May 17th, the girth at umbilicus measured $47\frac{1}{2}$ inches; but the head, neck, arms, and chest were free from œdema. The dyspnœa increased, and mucus much streaked with red blood was expectorated. The urine was very scanty, but was free from albumen, having been tested daily till May 17th. The patient gradually grew weaker, and in the evening of May 18th he became suddenly much worse. His pulse became very quick (136) and irregular: he was very restless and delirious, and his tongue was dry and brown; bowels relaxed. There was no increase of the dyspnœa, no lividity of the face, and no alteration in the physical signs presented by the lungs. The patient continued much in the same state until his death, at 11 a.m. of the following day.

On *post-mortem* examination, there was great lividity and puffy swelling of the face and neck, and discoloration of the integuments along the course of the subcutaneous veins. The heart was

healthy; there was no appearance of pleurisy or pneumonia, but both lungs were very congested and cedematous; the peritoneum contained several gallons of slightly turbid, straw-coloured serum. Notwithstanding the absence of albuminuria, both kidneys were much enlarged, the right weighing 9 ounces, and the left 9½ ounces. The capsules were non-adherent, and the surfaces smooth; the cortex was hypertrophied, flabby and soft, and mottled with numerous small, dark, ecchymotic spots. The renal epithelium was loaded with fine granules, but contained little oil. The spleen was large and soft, and weighed 7 ounces.

The liver was very small and quite hidden below the right ribs. It measured nine inches from right to left; the antero-posterior diameter of the right lobe was 6½ inches and of the left 5 inches; its weight was forty-three ounces and three-quarters, avoirdupois. The outer surface was nodulated and granular, and presented the typical characters of cirrhosis. The capsule was not thickened nor adherent. The structure of the organ was much increased in density, and on section presented islands of light yellow secreting tissue, the cells of which contained much oil, surrounded by broad bands of firm white tissue including the bile-ducts and hepatic vessels. The gall-bladder was distended with four ounces of thin, watery, greenish bile, in which were a large number of minute black concretions of inspissated bile, several of which were also found in the cystic, hepatic, and common ducts. The fæces in the intestines were coloured yellow.

Case LI. is another illustration of true cirrhosis arising from spirit-drinking. The cerebral symptoms were due to the complication of disease of the kidneys. The attacks of jaundice from which the patient suffered were probably the result of catarrh of the bile-ducts. The fact of their being preceded on each occasion for some days by vomiting pointed to antecedent irritation in the stomach and duodenum. That the jaundice was independent of the cirrhosis is shown by its almost disappearing before death, although the symptoms of portal obstruction increased.

CASE LI.—*History of Spirit-drinking—Cirrhosis of the Liver—Nephritis—Epileptiform Convulsions, and Death by Uræmia.*

Derby H—, aged 45, was admitted into the Middlesex Hospital, under my care, on October 15, 1867. He was a barnan, and for six or seven years he had been in the habit of drinking large quantities of gin, and during the last six months he had been very often intoxicated. About two years before admission he began to suffer from attacks of vomiting, followed after a few days by temporary jaundice, but not accompanied by pain, like that of biliary colic. Twelve months before admission the vomiting became more constant and urgent, and was accompanied by diarrhœa, and by a persistent pain in the region of the liver, with slight jaundice. The vomited matters often contained blood. He had also several attacks of epistaxis, one of which was so severe that he was taken to Charing Cross Hospital, where his nostrils were plugged. Six weeks before admission he had a severe fit of epileptiform convulsions, in which he bit his tongue deeply. In the following three weeks he had four similar attacks, the last of which was followed by jaundice, and a condition resembling delirium tremens, which continued up to the time of admission.

On admission, the patient's mind was very confused, his expression stupid, and he had considerable stupor. The conjunctivæ and the whole surface of the body were rather deeply jaundiced, and there was very slight pitting of the lower extremities. The tongue was dry and brown down the centre, and on the left margin, where it had been bitten in one of the fits, there was a deep ulcer. The motions contained bile. There was now neither vomiting nor diarrhœa, but there was evident tenderness on pressure below the right ribs. The liver could not be felt, and the hepatic dulness in the right mammary line was diminished, not exceeding three inches. At the margin of the ribs, however, corresponding to the situation of the gall-bladder, a distinct rounded tumour could be felt, about the size of a hen's egg. The abdomen was much distended from tympanitis, measuring at the umbilicus $32\frac{3}{4}$ inches; no sign of ascites could be discerned, but the splenic dulness was increased, and the subcutaneous veins of the abdomen were enlarged. The pulse was 84, feeble but regular; the cardiac dulness was slightly increased towards the left, measuring transversely $2\frac{1}{2}$

inches; the sounds were weak, but appeared to be free from any bellows-murmur. There was no dyspnœa or cough, and the physical signs of the lungs were normal. The urine was very dark, like porter; sp. gr. 1020; it contained abundance of bile-pigment, and a small trace of albumen. Under the microscope a few blood-corpuscles were seen, but no casts. The temperature was 101° 2.

The patient was treated with laxatives, diaphoretics, and diuretics, and a simple diet without any stimulants; he had also several warm baths, and mustard poultices applied to the back of his neck and over the region of the liver. Under this treatment, after two or three days, he began to improve, and at the end of ten days he was able to get up and go about the ward. The jaundice and tympanitis were greatly diminished; the tumour in the region of the gall-bladder had disappeared, the tongue was moist and clean, the appetite had returned, and the temperature was normal. The urine, however, still contained a slight trace of albumen ($\frac{1}{20}$), and the patient's memory was confused as to dates. With the exception of a painful inflammatory swelling in the meatus of the left ear, lumbar pain, an attack of vomiting with slight epistaxis on November 22, and a return of the stupor with dryness of the tongue during the last week of November, this improvement lasted until December 12. About this time he became much weaker, and very restless and delirious at night; his abdomen had again become enlarged, measuring at the umbilicus $35\frac{1}{4}$ inches, and there was now unmistakeable evidence of a small quantity of fluid in the peritoneum. There was also considerable œdema of the lower extremities, and a distinct systolic blowing murmur at the base of the heart. The albumen in the urine, however, was not increased, the temperature was only 96° F., and there was scarcely any jaundice. The treatment consisted in the administration of liquor ammoniæ acetatis with acetate of potash, tincture of digitalis, and decoction of broom-tops with mild laxatives, and subsequently compound jalap powder, and croton oil, and the application of mustard and linseed poultices to the loins.

The ascites, however, gradually increased until the abdomen measured 40 inches, but after the patient kept his bed the dropsy of his legs almost disappeared, and there was no trace of dropsy of the face or upper part of the body. The enlargement of the abdominal veins increased. The urine was scanty and smoky, and it contained a much larger amount of albumen ($\frac{1}{6}$), with

epithelial and blood casts. The tongue was dry and brown; the motions and urine were passed in bed, but the temperature throughout was rather below the normal standard. For three or four days before death, which took place on December 23, there was much noisy incoherent delirium with spasmodic twitchings of the extremities, but no general convulsions.

The appearances found after death were as follows:—Slight jaundice of the integuments. Several quarts of clear straw-coloured serum in the peritoneum. Liver very small, measuring only 9 inches from right to left, and 6 inches from back to front, in right lobe: greatest thickness $3\frac{1}{4}$ inches; weight 4½ ounces; outer surface coarsely granular; glandular substance extremely dense, and consisting of firm bands of fibrous tissue, including bile-ducts and obliterated vessels, and enclosing islets of yellow secreting tissue, the cells in which were loaded with yellow pigment and oil. Spleen large, 7 ounces, firm. Pancreas very large and indurated from the presence of an unusual amount of fibrous tissue. Extreme catarrhal inflammation, with hæmorrhagic erosions of mucous membrane of stomach. Both kidneys enlarged, weighing together 14 ounces; capsules separating readily; surfaces smooth; cortices hypertrophied; marked injection of straight vessels in pyramids and of Malpighian bodies; renal tubes gorged with granular epithelium. Considerable hypertrophy of left ventricle of heart, and a vegetation the size of a hemp-seed, on the ventricular surface of each of the aortic valves. Much hypostatic congestion of both lungs, and a few old adhesions over surface of left. Much serous fluid containing urea beneath the arachnoid and in the lateral ventricles of the brain.

In the following case, there was every reason to believe from the history that the atrophy of the liver was due to true cirrhosis, but the only indications of portal obstruction, while the patient was under observation, were copious hæmatemesis, hæmorrhoids, and slight enlargement of the spleen. In several instances, however, I have known persons die suddenly of hæmatemesis, who had previously been sufficiently well to perform their duties in life, and in

whose bodies well-marked cirrhosis of the liver has been found after death.

CASE LII.—*History of Spirit-drinking—Contracted Liver—Copious Hæmatemesis—Delirium Tremens.*

Eliza D—, aged 29, a person in good circumstances, was admitted into the Middlesex Hospital, under my care, on February 5th, 1867. She had been married for five years, and was the mother of two children. Ever since her marriage, and perhaps before, she had been addicted to spirit-drinking. This had led to a separation from her husband. For a long time she had been in the habit of getting drunk two or three times a week. She had not suffered, however, from pain and vomiting after food.

Early in the morning of the day of admission, after a restless night, the patient vomited matter streaked with blood, and an hour afterwards she brought up a large quantity of pure blood. The medical man who was called to see her, said that there were at least two pints. Brandy and ice were administered, but she vomited more blood, and on trying to get up, she fell down in an insensible state, and was brought to the hospital at noon.

On admission, the skin was noted to be sallow, although there was no decided jaundice. There was tenderness at the epigastrium and the hepatic dulness was diminished, not amounting to 3 inches in the right mammary line; the splenic dulness was increased, and there were several small hæmorrhoids about the anus, but there was neither ascites nor enlargement of the abdominal veins. The urine contained a trace of albumen, and there was a faint systolic bellows-murmur over the heart, but the cardiac dulness and impulse were not increased, the physical signs of the lungs were perfectly normal, and there was no œdema of the legs.

The patient was treated with gallic acid and opium, and with ice and milk. For several days the vomiting continued, but, except on the day of admission, the vomited matters contained no blood. The bowels did not act for five days after admission; enemata then brought away a large quantity of tar-like matter. For several days after admission the patient suffered from a severe attack of delirium tremens, but by February 11 this had quite subsided, and she was able to eat and retain solid food. The vomiting did not recur, and on February 16, she was discharged.

Many of you watched the patient, whose case I am now about to relate, with much interest, and it is to be regretted that no opportunity was afforded for examining the condition of his liver after death, inasmuch as there was considerable obscurity as to the cause of the atrophy. The signs of obstructed portal circulation, however, were well-marked, and the circumstance of hæmatemesis preceding the other signs of obstruction, for several years, is interesting in connection with what was observed in Cases L. and LII.

CASE LIII.—*Chronic Atrophy of the Liver—Ascites—
Hæmatemesis and Bloody Stools.*

James T——, aged 38, was admitted into the Middlesex Hospital, under my care, on August 20th, 1866. For 6 years he had been a brewer's drayman, and been accustomed to drink a good deal of ale, but not spirits; before that he had been a farm labourer and had drunk little of alcohol in any shape. He had never suffered from ague or rheumatic fever, but at the age of 18 he had been laid up for a year with a cough and debility, and had been told at the Reading Infirmary that he had consumption. He recovered, however, and remained well until 8 years before admission, when he received a kick from a horse on the right side. He did not take much notice of this at the time, and followed his work for 5 or 6 weeks afterwards. Whether owing to this injury or not, he then began to suffer from great pain and tightness at the epigastrium, with constipation. He took some aperient medicine, which operated, but on the following day he vomited a large quantity of clotted blood, and for a week afterwards he continued to pass blood per anum. This left him very weak, but relieved the pain, and he returned to his work. After this he had a similar attack about once a year, the only difference being that the quantity of blood lost was less than it had been the first time. On each occasion the vomiting of blood had been preceded for several days by great headache, nausea, and pain in the abdomen.

The last attack had occurred four months before admission. In the spring of 1865 he had been for several weeks in a metropolitan hospital for the hæmorrhage. Shortly after leaving the hospital, in April, 1865, his abdomen became swollen, and subsequently, his legs. He drank 'broom-tea,' and the swelling subsided, but a month before admission it increased again.

On admission, the patient was emaciated and yellow, but the conjunctivæ were white. The abdomen was greatly distended from the presence of fluid in the peritoneum, and the veins of the abdominal wall were unusually large and distinct, but there was nowhere tenderness, except on pressure over the right hypochondrium. The liver could not be felt, and the hepatic dulness in the right mammary line measured only $2\frac{1}{2}$ inches, an observation which was subsequently confirmed after paracentesis. The tongue was slightly furred; the bowels were costive; and there was slight flatulence after meals, but the appetite was good. There was an anæmic bellows-murmur over the sternum, but the cardiac dulness was not increased. The respirations were 20 and easy; at the bases of both lungs there was a little fine crepitation. Upwards of 40 ounces of urine were passed daily; it was dark, but contained neither albumen nor bile-pigment. There was moderate œdema of both legs.

The treatment consisted in purgatives and diuretics, and for some time bromide of potassium in five-grain doses three times a day. A generous diet was allowed, but no stimulants.

At first there was considerable improvement, and the girth of the abdomen was reduced 2 inches; but about the middle of September the swelling began to increase again, and on the 28th the abdomen measured 42 inches, the integuments over it were tight and glistening, and the urine was reduced to about a pint daily; the respirations were 32, and considerably embarrassed. On September 30th, the patient suffered much from pains in the abdomen, and during the following night he began to pass frequent motions containing much black blood. On October 6 the patient had still diarrhœa with bloody stools; the abdomen had increased to 44 inches; the legs also were swollen, and there was great orthopnoea. Paracentesis was performed, and 17 pints of fluid were drawn off. The fluid was clear, straw-coloured, and alkaline; its specific gravity was 1012, and it contained a large quantity of both chlorides and albumen. The operation gave great relief to the

breathing; the anasarca of the legs diminished; the urine rose to two pints; and the blood disappeared from the motions.

Two days, however, had not elapsed before the swelling was again noticed to be increasing, and on October 15th the abdomen measured 42 inches, the cedema of the lungs had extended, and the patient suffered much from dyspnoea and a troublesome cough. On October 21 vomiting came on, and the vomited matters contained a good deal of blood; the motions also again contained blood. These symptoms continued until October 24th, when the patient insisted on leaving the hospital. He was removed to Egham, where he died the same evening. His friends would not permit his body to be examined.

The two livers which I now show you, appear to me to account for certain differences of opinion still entertained respecting the pathology of cirrhosis. One was taken from the case I have already detailed to you, as a good example of true cirrhosis (Case L.), the other from the body of the patient whose case I am about to mention to you (Case LIV.).

On the one hand, it is stated that in cirrhosis there is an increase of fibrous tissue, the result of a chronic inflammatory process, and that the secreting tissue becomes atrophied from the pressure exerted on it by this fibrous tissue, or from the conversion of the gland-cells into fibre-cells; while, on the other, it is contended that the secreting tissue is simply atrophied, and that the fibrous tissue is not absolutely, though relatively, increased. The former view is the one advocated by Dr. Budd,* in his excellent work on Diseases of the Liver, and the latter has been put forward by Dr. Beale,† who has found the interlobular tissue in cirrhosis intimately permeated

* Third ed. p. 136.

† Archives of Med. vol. i. p. 125.

by blood-vessels and minute bile-ducts. A third class of pathologists, among whom may be mentioned Förster, believe that there are two forms of granular cirrhosis, one in which the fibrous tissue is increased, and another where it is not, and this is the view which I have already placed before you (see pages 239, 240, 252). In one of these two livers the structure is extremely dense, and the fibrous tissue appears greatly increased, not only to the naked eye, but on microscopic examination; while in the second liver, although the atrophy is extreme, so that the weight is little more than one-half that of the first, the tissue is extremely soft and friable, and there is no evidence of any increase of the fibrous tissue, either to the naked eye, or on microscopic examination. If the increased density and apparent increase of fibrous tissue in the former case be due merely to the disappearance of a portion of the secreting tissue, it would be difficult to account for the fact that in the second case, although the atrophy is much greater than in the first, the consistence of the organ is much less than in health, and there is no apparent increase of the fibrous tissue. Both patients exhibited during life the ordinary phenomena of portal obstruction met with in cirrhosis; but there was this difference between the two, that the patient with the dense fibrous liver had led a very intemperate life, whereas there was no history of intemperance in the other patient. I am unable to throw any light on the etiology of the disease in the latter case; but

the absence of a history of spirit-drinking, which is almost universal in the dense fibrous cirrhotic liver, is worth noting.

CASE LIV.—*No history of Spirit-drinking—Symptoms of portal Obstruction—Soft, atrophied, granular Liver—Spurious Cirrhosis.*

M. D.—, aged 63, was admitted into the Middlesex Hospital, under my care, on the 1st of April, 1867. Her health through life had, on the whole, been good, except that at the age of 45, she had been confined to bed for six weeks, with what she believed to have been rheumatic fever. Since then she had not suffered from either dyspnœa or palpitations; her habits had always been temperate. Her present illness commenced six weeks before admission, with vomiting and purging. Everything she swallowed was rejected within ten minutes; these symptoms continued, and after three weeks it was first noticed that she was slightly jaundiced, and about the same time she passed a good deal of blood from the vagina.

On admission, the patient was thin and very weak; she had a well marked arcus senilis, and decided jaundice of the skin, conjunctivæ and urine. The pulse was 108, and very irregular. There was visible pulsation of many of the arteries, which felt rigid and tortuous. The impulse of the heart was strong but irregular, and the cardiac dulness was slightly increased, but no bellows-murmur was audible. The respirations were 28 and rather laboured; coarse moist râles were audible at the bases of both lungs. The abdomen was considerably distended and tympanitic, measuring 33 inches in circumference at the umbilicus; but there was no distinct indication of the presence of fluid in the peritoneum, and no enlargement of the subcutaneous veins of the abdomen. There was considerable œdema of both lower extremities, but the urine contained no albumen. The tongue was moist, with a white fur: the motions passed after admission were dark brown, and contained abundance of bile.

On April 6, the vomiting and diarrhœa had subsided, but there was tolerably clear evidence of fluid in the peritoneum, and slight enlargement of the subcutaneous abdominal veins. The girth at the umbilicus was nearly $35\frac{1}{4}$ inches; but it never exceeded this.

From this date the patient continued in a very low state, but without any increase of the abdomen or, indeed, change of any sort, until April 30, when the vomiting returned, but not the diarrhœa. The patient now lost all appetite; her tongue became dry and brown; and she continued to sink until her death, on May 13. For the last twenty-four hours of life she was quite unconscious.

On *post-mortem* examination, both kidneys were found to be contracted and granular, with numerous cysts in their cortical substance. There was considerable hypertrophy of the left ventricle of the heart, and atheroma of the aorta, but the valves were healthy. The lungs were slightly emphysematous, but otherwise normal. The peritoneum contained about a gallon of clear straw-coloured serum. The intestines had a fleshy appearance (from maceration) and there was slight ecchymosis in the mucous membrane of the cæcum, but in other respects they were normal. The spleen was of natural size; with the exception of a small fibrous tumour, the uterus also was healthy. •

The liver was extremely small, weighing only 25·5 ounces avoird., and measuring 7·75 inches from right to left, 5·5 inches antero-posteriorly in right lobe, and 4·75 in left. Its capsule was not at all thickened, and was not adherent; but its outer surface was coarsely nodulated and granular, exactly as in cirrhosis. The margin of the organ all round, but particularly in front, had a winged appearance, from the total disappearance of the secreting tissue between the capsule on the upper and under surfaces. At the anterior margin of the right lobe this attenuated rim was nearly an inch in width, and only about a third of an inch thick. On section of the organ, there was no evidence of any increase of the fibrous tissue; on the contrary, the consistence was extremely soft. The cut-surface presented a yellowish-brown colour, and a coarsely granular appearance, from the aggregation of the lobules into small masses. The outline of the individual lobules was not well defined; but on microscopic examination the secreting cells were found in abundance, though loaded with oil; there was no leucine or tyrosine. The attenuated rim presented a smooth grey appearance on section, and was made up for the most part of fibrillated tissue and vessels, with here and there a few collapsed secreting cells. Projecting from this rim were a few isolated nodules, of yellowish-brown hepatic tissue, about the size of peas.

Case LV. is an instance of chronic atrophy of the liver, arising from peri-hepatitis. The appearance of the organ is similar to what is often seen in constitutional syphilis, although no evidence could be made out of the patient having suffered from syphilis. The liver was very small, yet there was no indication of portal obstruction; but in another patient, whose body I dissected some years ago, there was a similar condition of liver with great ascites. The spleen weighed 27 oz., and for three days before death there had been severe vomiting and purging, with much blood in the vomited matter and stools.

CASE LV.—*Chronic Atrophy of the Liver, from Perihepatitis—Simple Ulcers of the Stomach.*

The liver and stomach which I now show you were removed by me from the body of a woman, aged 44, who died in this hospital some years ago, under the care of Dr. Thompson. She was admitted on March 19, and died on April 15, 1861. Six months before admission she began to get thin, and to suffer from nausea and loss of appetite, and six weeks before admission pain and vomiting after food came on. The symptoms noted, while the patient was under observation, were great emaciation, tenderness in the region of the liver, the dulness of which measured less than 2 inches in the right mammary line, pain and vomiting after food, and constipation. There was no jaundice, no ascites, and no albumen in the urine; the heart's sounds were normal.

After death the liver was found to weigh only $30\frac{1}{2}$ ounces; it was very small, its dimensions being—extreme length, 9 inches; ant. post. diameter, 6 inches; greatest thickness, 2 inches. The capsule was thickened, and was connected to the diaphragm and ribs by numerous fine long fibrous bands. Its outer surface was marked by extensive cicatrix-like depressions, and scattered through its substance were many firm fibroid nodules, about the size of a pea, composed of fibrillated tissue with oily and granular matter. The glandular tissue which remained appeared healthy. The

pyloric end of the stomach was thickened and narrowed from what appeared to be the cicatrices of former ulcers. Two inches from the pylorus there was an open ulcer the size of a threepenny piece. The spleen was not enlarged. There was commencing waxy disease of the kidneys. There were a few small patches of recent lobular pneumonia in both lungs; the base of the right lung was connected to the diaphragm by fine adhesions. No cicatrices could be discovered on the labia, in the groins, or over the tibæ.

In Case LVI. the atrophy of the liver appeared to be secondary to disease in the chest.*

CASE LVI. — *Bronchitis and Dilated Bronchi — Disease of the Aortic Valves—Contracted Liver—Great Ascites.*

The liver which you see here is not much more than one-half of the normal size; it weighed only 33 ounces. Its outer surface is finely granular, being marked by numerous small depressions, corresponding to the centre of the lobules. The capsule at many places is much thickened, and was adherent to the surrounding parts, and the fibrous tissue in the interior of the organ is increased. Before immersion in spirit, the surface on section presented a nutmeg appearance.

This liver was taken from the body of a man aged 40, who was a patient in this hospital from June 27 to July 16, 1860, and again from November 13, 1860, until his death, on January 5, 1861. He had been in the habit of drinking spirits, but not in excess. His illness commenced about a year before death with cough, dyspnoea, and other signs of bronchitis. After three months his legs began to swell, and subsequently his abdomen, but at the time of his first admission the anasarca of the legs was comparatively slight, although the abdomen was enormously distended from ascites. The patient suffered much from pain below the right ribs; the hepatic dulness was diminished; there was no albumen in the urine, but there was a diastolic blowing murmur at the base of the heart.

The bronchial tubes after death were found to be much thickened and dilated, and the pulmonary tissue at many places was in a state of fibroid degeneration. The right lung was inseparably

* See also the case of Mary T——, related in Lecture XI.

adherent to the wall of the chest. The right cavities of the heart were dilated; the aortic valves were incompetent; two of the flaps were united into one, and in all of them there was a considerable amount of atheroma.

The following case was published by me some years ago, in the 'Pathological Transactions,' vol. vii. p. 238. It was an interesting example of chronic atrophy of the liver, in conjunction with great enlargement of the spleen and leukæmia. Although the organ was described at the time as 'in an advanced stage of cirrhosis,' the firm adhesions of the liver, omentum and spleen, the thickened capsule of the spleen and the obliteration of the cystic duct, all pointed to chronic peritonitis as the probable cause of the atrophy. There was no history of spirit-drinking. The remarkable circumstance, however, which induces me to mention the case is that on four different occasions large quantities of fluid were abstracted from the abdomen by paracentesis, and that the patient lived for nearly two years afterwards without any reaccumulation.

CASE LVII.—*Chronic Atrophy of the Liver and Ascites—Paracentesis—No Accumulation after fourth Tapping—Enlarged Spleen and Leukæmia—Death from Ulceration of the Mouth and Pharynx, and Necrosis of the Jaw and Vertebrae.*

The patient was a female, who had been born and always resided in London. She had been very temperate, but always very delicate. The catamenia had not appeared until she was twenty, and although married for eleven years, she never had any children or miscarriages. In 1850, when 31 years of age, she first noticed

a swelling below the right ribs: but this did not give her much inconvenience till the end of 1853, when the whole abdomen began to enlarge, and on April 12th, 1854, she was admitted into the Hospital for Women, in Soho Square, under Dr. Tanner.

She was then suffering from symptoms of diseased liver and ascites, and her abdomen measured $43\frac{3}{4}$ inches in circumference, and $18\frac{1}{2}$ inches from the ensiform cartilage to the pubes. During her stay in hospital she was treated with iodide of potassium, mercurial ointments and purgatives, and the abdomen was tapped four times. On April 17th, 356 fluid ounces of clear fluid were drawn off; on May 19th, 400; on June 16th, 431; and on July 7th, 404; altogether 1591 fluid ounces. After the last operation, the fluid did not collect again, and the patient left the hospital greatly improved in health, and with the abdomen of the natural size.

There was no return of the ascites; but some months afterwards she began to suffer from ulceration of the mouth and throat, producing a very fetid discharge. Several of her teeth came out, and in October, 1855, a portion of the alveolar process of the lower jaw exfoliated. She had also several severe attacks of epistaxis, and bleeding from the gums. On March 12th, 1856, she came under my care. She was then extremely weak and confined to bed. There was extensive ulceration of the fauces and along the margin of the gums, but the voice was natural. She stated that she had never suffered from syphilis, nor taken mercury internally. The whole of the left side of the abdomen was filled up by a solid tumour, extending forwards to within $2\frac{1}{2}$ inches of the umbilicus, but there was no ascites, and the hepatic dulness was diminished, not exceeding $2\frac{1}{2}$ inches in the right mammary line.

The ulceration of the mouth rapidly extended. More teeth and pieces of bone came away from the jaw; the body of one of the cervical vertebræ became exposed; and the dysphagia was so great that at last even fluids were rejected by the nares.

After death, less than a pint of clear serous fluid was found in the abdominal cavity, and the omentum was firmly adherent to the abdominal wall. The liver was very small, and weighed only 35 ounces. Its outer surface was firmly adherent to the surrounding parts, and the organ was described at the time as 'in an advanced stage of cirrhosis.' There was bile in the gall-ducts and intestines, but the gall-bladder contained none. The cystic duct was obliterated, and the vessels and duct in the portal fissure passed through a quantity of firm fibrous tissue; the gall-bladder

was collapsed ; its lining membrane was perfectly white and encrusted with small fragments of earthy matter, some of which were firmly adherent. This consisted mainly of carbonate of lime ; it effervesced with acids, and microscopic examination showed it to be made up of rounded crystalline particles, varying in size from $\frac{1}{400}$ of an inch to an almost infinite degree of minuteness ; some of them seemed to be made up of radiating acicular crystals cohering in the centre (fig 22).

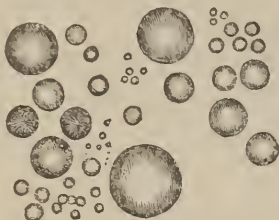


Fig. 22. Minute crystalline masses of Carbonate of Lime from Gall-bladder in Case LVII., magnified 180 diameters.

The spleen weighed $68\frac{1}{2}$ ounces, and measured $11\frac{3}{4}$ inches by 4 inches. Its capsule was much thickened, indurated, and adherent



Fig. 23. Microscopic appearances of the Blood in Case LVII., magnified 400 diameters.

to surrounding parts. The splenic tissue was firm and of a reddish brown colour, mottled with numerous lighter specks like sago-grains. The blood from all parts of the body exhibited the appearances

described by Virchow under the designation of leukæmia. There was a great increase of the colourless corpuscles which, on the addition of weak acetic acid, presented a single, double, treble, or quadruple nucleus. Some of these nuclei were crescentic, and many of them appeared depressed in the centre like small red corpuscles. Some of the cells, in addition to the nuclei, contained a few minute oil-globules, and there was also a considerable quantity of free granular matter and oil-globules. The red corpuscles showed no unusual tendency to collect in rolls as in some cases of leukæmia (see fig. 23).

The heart, lungs, and kidneys were healthy.

LECTURE VIII.

JAUNDICE.

DEFINITION — IMPORTANCE OF RECOGNIZING CAUSES — PHENOMENA OF JAUNDICE: 1. LOCALITIES; 2. SECRETIONS; 3. BITTER TASTE; 4. DERANGEMENTS OF DIGESTION; 5. ITCHINESS; 6. CUTANEOUS ERUPTIONS; 7. TEMPERATURE; 8. PULSE; 9. HÆMORRHAGES; 10. GENERAL DEBILITY; 11. YELLOW VISION; 12. CEREBRAL SYMPTOMS.—THEORY OF JAUNDICE.

JAUNDICE may be defined as a yellowness of the integuments and conjunctivæ, and of the tissues and secretions generally, from impregnation with bile-pigment. The word jaundice in fact is derived from the French *jaune*, yellow. The technical name *icterus* is less appropriate, being the Greek word for the golden thrush, a bird with golden plumage, the sight of which by a jaundiced person was believed by the ancients to be death to the bird, but recovery to the patient.

Few morbid symptoms are due to such multifarious causes as jaundice, and there are none in which it is more difficult or important to determine the cause. It is too much the custom for even medical men to be satisfied with the fact that a patient has got 'jaundice,' and to administer remedies which are supposed to exercise some special action on the liver, without taking much trouble to investigate further;

and yet the prognosis and whole treatment of the case ought to depend, not on the jaundice, but on its cause. The difficulty of the subject is further increased by the fact that even those who have devoted most attention to the subject are not at one as to the mode of production of jaundice in many cases. I shall endeavour in these lectures to explain to you the various causes of jaundice, the mode in which they operate, and the means of distinguishing them. But in the first place it will be well that I should refer to instances of what may be called *spurious* jaundice, and describe to you certain phenomena connected with jaundice irrespectively of its cause.

SPURIOUS JAUNDICE.

In the first place, then, you must be quite certain that you have to deal with jaundice in any case, before proceeding to investigate its cause. As a rule there is no great difficulty in the diagnosis ; you have only to look at the patient to know what is the matter with him. There are, however, certain other conditions which are apt to be mistaken for the slighter forms of jaundice, and it is very necessary that you should keep in remembrance these sources of fallacy.

1. First, there is the greenish-yellow colour of countenance observed in the anæmic state known as *chlorosis*, and which is due to a morbid alteration of the blood. This is distinguished by—

a. A pearly whiteness of the conjunctivæ, and pallor of the lips and tongue.

b. Other symptoms of anæmia, such as a feeble pulse, and a systolic bellows-murmur at the base of the heart, without any other indication of disease of the heart or of leukæmia.

c. In females, amenorrhœa, or some other indication of uterine derangement.

d. Absence of bile-pigment from the urine.

2. There is a peculiar greyish-yellow waxen tint of skin characteristic of organic visceral disease, and especially of cancer. This is distinguished from jaundice by—

a. The absence of any yellow tint of the conjunctivæ.

b. The absence of bile-pigment from the urine.

c. The presence of other symptoms of visceral disease, or in the case of cancer of the cancerous cachexia.

3. A dusky yellowish colour of the surface is not unfrequently developed in persons who have suffered long or often from malarious fevers, and sometimes also in those whose systems have been poisoned by lead. This condition is to be recognized by—

a. The absence of any yellow tint of the conjunctivæ.

b. The absence of bile-pigment from the urine.

c. The fact of the individual having suffered from malarious fevers, or lived in a malarious country.

d. Exposure to the poison of lead, with a blue line along the margin of the gums, or a history of lead-colic or palsy.

4. A yellowish colour of the conjunctivæ may be due to sub-conjunctival fat. This is distinguished by—

a. The yellow tint not being uniform.

b. The absence of jaundice of the skin, or of bile-pigment in the urine.

5. In a large number of the cases of the so-called jaundice of new-born children (*icterus neonatorum*), the yellow colour which appears on the third or fourth day after birth is not due to jaundice at all, but is merely the result of changes in the blood in the over-congested skin, the vivid redness of the new-born babe fading as bruises fade, through shades of yellow into the genuine flesh colour.* Young infants, however, are liable to real jaundice, to which I shall refer hereafter. The spurious affection differs from this in—

a. The conjunctivæ being of the natural colour.

b. The urine being free from bile-pigment.

c. The gradual fading of the yellow colour of the skin after a few days.

d. The child being quite well, and the bowels acting properly.

6. The bronzing of Addison's disease is not likely to be mistaken for jaundice. It differs—

a. In the browner or more dusky character of the discoloration, and in the fact of its being darker at certain parts, such as the face, neck, hands, areolæ of the nipples, axillæ, penis, scrotum, &c.

b. In the presence of other symptoms of Addison's

* See West, *Dis. of Children*, 5th ed. 1865, p. 601.

disease, and particularly of extreme anæmia and vomiting.

c. In the whiteness of the conjunctivæ.

d. In the absence of bile-pigment from the urine.

7. Persons who have been much in hot climates or exposed to the sun may have a permanent bronzed appearance of the face, which is distinguished from jaundice by—

a. The skin of the chest and other parts of the body having a natural tint.

b. Whiteness of the conjunctivæ.

c. Absence of bile-pigment from the urine.

8. Other pigments in the urine may give to this secretion a colour which may be mistaken for that of bile-pigment, such for instance, as those which are common in diseases interfering with the respiratory functions. But the bile-pigment can always be recognized by the urine staining the linen yellow, and still better by testing with nitric acid. If you pour a small quantity of urine containing bile on a white plate, or on a sheet of writing paper, and carefully allow a drop or two of nitric acid to fall upon it, an immediate play of colours will be produced around the spot where the acid falls, passing from brown through green, blue, violet, and red, into a dirty yellow.*

* According to Frerichs this reaction may fail in consequence of the bile-pigment in the urine having undergone some transformation, in cases where the other symptoms of jaundice are undoubted. When this is the case, the urine is at one time of a brown or brownish-red colour, and becomes red on the addition of nitric acid; at another time

9. Lastly, those of you who may enter the public services ought to remember that jaundice has been successfully feigned by soldiers and sailors desirous of obtaining a discharge. The yellow colour of the skin has been simulated by painting it with infusions of saffron, turmeric, rhubarb, broom-flowers, or soot, while the colour of the urine has been heightened by taking rhubarb or santonine.* But in feigned jaundice you will find—

a. That the conjunctivæ are white.

b. That bile-pigment cannot be detected in the urine by the nitric-acid test; and,

c. That soap and water, or better still, a weak solution of chloride of lime, will remove the yellow colour from the skin.

d. If the urine be coloured by the use of santonine or rhubarb, it will be rendered blood-red by the caustic alkalies or their carbonates.

In the next place I will call your attention to—

CERTAIN PHENOMENA AND SYMPTOMS OF JAUNDICE IRRESPECTIVE OF ITS CAUSE.

1. The *Locality and Intensity* of the jaundice. Most of the organs and tissues of the body become impregnated with bile-pigment. This first accumulates in

it is of a deep red, which is converted by nitric acid into a dark bluish-red. (Dis. of Liver, Syd. Soc. Ed. i. 100.) I have made a similar observation in rare cases where jaundice has resulted from a blood-poison, and I have frequently found the urine to present these characters where there has been no jaundice, but obvious derangement of function, or alteration of structure, of the liver.

* Gavin, on Feigned and Fictitious Diseases, 1843, p. 389.

the blood, and the jaundiced tint penetrates every part of the body that is permeated by blood—even the brain, the bones, and the foetus in utero. The mucous membranes, however, are but slightly coloured, although the tongue is often distinctly yellow. In cases of intense jaundice even the humors of the eye assume a yellow tinge.

The intensity of the jaundice varies in the different tissues of the body. When the jaundice depends on obstruction of the common bile-duct, the liver itself is the organ that is most deeply coloured; it often presents a deep olive-hue. But when there is no impediment to the flow of bile into the bowel, the liver may not be more jaundiced than other parts.

Next to the liver, the skin is the tissue which becomes most deeply jaundiced; but before it becomes affected a yellow tint is usually observed in the conjunctivæ. There must be a certain concentration of bile-pigment to produce a yellow colour of the skin; in the slighter and more temporary cases, the conjunctivæ only may be affected. Even the conjunctivæ may not become yellow for two or three days after the cause of the jaundice has been in operation. It has been shown by experiment that after the passage through the ducts has been arrested, two or three days will often elapse before the conjunctivæ become jaundiced.

The colour of the skin varies from a pale sulphur or lemon-yellow, through a citron-yellow, to a deep olive or bronzed hue. The tint varies according to the cause and the duration of the disease. When

the cause is obstruction of the bile-duct, it is light at first, and increases in depth the longer the disease lasts. When the jaundice is independent of obstruction to the flow of bile, the colour is rarely very deep, and yet these are often the most serious cases. Instances occur where the jaundice is of a greenish or almost black hue, owing to the bile-pigment which is absorbed being vitiated and dark, or the visage being already livid, from imperfect arterialization of the blood, the green colour being a result of the mingling of the blueness of lividity with the natural yellowness of jaundice. The colour also varies with the age, the natural complexion, and the amount of fat in the individual. It is deeper in the old, the wrinkled, and the dark-complexioned, than in young persons of fair complexion, and with plenty of fat. Lastly, it is important to remember in reference to treatment that the colour often remains in the skin for some time after the cause of the jaundice has been removed, and that then its departure may be expedited by diaphoretics and warm-baths.

2. *The Secretions* are tinged with bile-pigment, but some much more so than others. This is notably the case with the urine, by which the greater part of the bile-pigment in jaundice is eliminated from the body, and which acquires a saffron-yellow, greenish-brown, or brownish-black hue, according to the amount of pigment which it contains. The urine usually becomes yellow before there is any yellow tint of the skin, or even of the conjunctivæ, and it may happen,

when the cause of the jaundice is temporary, that the whole of the pigment is eliminated by the urine, without any jaundice appearing in the skin. On the other hand, as I have already informed you, when once the skin becomes yellow, it may remain so for some time after bile-pigment has quite or nearly disappeared from the urine.

The precipitates which fall from jaundiced urine sometimes contain renal epithelium, or casts of the renal tubes tinged yellow.

Other secretions, in cases of jaundice, contain bile-pigment as well as the urine.

The cutaneous glands usually eliminate the pigment and sometimes in such quantity as to stain the linen yellow, but the amount discharged in this way is small when compared with that which escapes through the kidneys.

Dr. Bright* and others have recorded instances where the secretion of the mammary glands has been found tinged with bile-pigment, but cases of this sort are not very common; still rarer instances have been noticed where the saliva, or the tears, have been similarly affected. It is not a little remarkable, however, that, notwithstanding statements of a contrary nature which have been made by Fourcroy,† and Dr. Osborne, of Dublin,‡ bile-pigment is not eliminated in cases of jaundice by the mucous membrane of the

* Guy's Hosp. Rep. 1st Ser. i. 623. See also Budd, Dis. of Liver, 3rd ed. p. 470.

† Frerichs, Op. cit. i. 103.

‡ Dublin Journal of Med. Feb. 1853.

respiratory passages, or of the digestive tube. This is a matter of some practical importance, for were the fact otherwise, the stools might contain bile-pigment even when there was complete obstruction of the gall-duct. Still when either of these mucous membranes is inflamed and separates an albuminous or fibrinous exudation from the blood, the altered secretions may contain bile-pigment. Thus when pneumonia coexists with jaundice, there is often bile-pigment in the sputa, which may be distinguished by the nitric-acid test from the greenish or yellow colour often presented by pneumonic sputa from changes in the blood-pigment independent of bile. Indeed, in cases of jaundice, bile-pigment may be detected in inflammatory exudations, as in the serum of a blister, before it appears in either the skin or even the urine. It is probable that those rare cases where the saliva has been noticed to be yellow admit of a similar explanation. In the cases recorded by Huxham* and Budd,† which have been so often referred to, there was mercurial salivation, in which condition the saliva is not normal, but contains much albumen.

3. A *bitter taste* is not unfrequently complained of by persons who are the subjects of jaundice. Sometimes this appears to be due to eructations from the stomach of bilious matter, but when the bile-duct is obstructed this of course is impossible. It then probably denotes the presence in the blood of the biliary

* Op. Physico-medica, tom. iii. p. 12.

† Budd. Op. cit. p. 469.

acids, for bile-pigment is tasteless, while taurocholic acid is intensely bitter.

4. *Derangements of Digestion.*—The chief derangements of digestion, resulting from the absence of bile from the intestines, are flatulence, constipation, and an altered character of the motions. Bile is endowed with powerful antiseptic properties, and consequently when it is absent, the intestinal contents undergo decomposition, gases accumulate in the bowels and cause tympanitic distention of the abdomen, and the motions acquire a putrid odour. Owing to the absence of bile-pigment also the motions present a pale drab or clay colour. Bile appears also to be the natural stimulant of the peristaltic action of the gut, and consequently when the supply is cut off the bowels are usually constipated. The putridity and paleness of the motions and the constipation, however, are confined to those cases where there is complete obstruction of the ducts. When the ducts are free, or where the obstruction is incomplete, and when bile still enters the bowel, the motions may be but little altered, and may be voided regularly.

When bile does not enter the bowel, the digestion of fat is interfered with. Jaundiced patients dislike fat, and do not assimilate it, the fatty matter in the ingesta being discharged with the fæces. This is still more remarkably the case when the pancreatic secretion is also prevented entering the bowel, but it was long since shown by Drs. Bright and Owen Rees*

* Guy's Hosp. Reports 1836, Ser. 1, vol. i. p. 610.

that in most cases of very obstinate jaundice, when there is complete obstruction of the bile-duct, an unusual quantity of fat may be detected in the stools. In all cases of jaundice therefore the nutrition of the body suffers: the emaciation may be slow, but it is usually progressive, until the fat disappears from the body, and then the weight of the body may remain stationary. Cases, it is true, have been recorded where patients have lived for several years with jaundice, and where there has been comparatively little wasting, but these cases are exceptional.

5. *Itchiness of the Skin* without any eruption is occasionally a very obstinate and distressing symptom in jaundice. In two cases the late Dr. Graves observed this itchiness precede the jaundice—in one for ten days, and in the other for two months—and cease as soon as the jaundice appeared. More commonly, it is first noticed at the commencement of the jaundice (Case LXIV.); sometimes it comes and goes, and at others, it persists as long as the jaundice lasts, being usually worse at night, and preventing sleep. You have now an opportunity of witnessing the great distress which this symptom may occasion in the case of William M——, now in Cambridge Ward (Case LXIV.). This man has had jaundice from obstruction of the common bile-duct for upwards of six months, and throughout has suffered from intense itchiness, which, notwithstanding opium, subcutaneous injections of morphia and anodynes of every sort, has caused him wretched nights. The

bicarbonate of potash is the only remedy that has appeared to give any relief. It is not known on what ingredient of the bile this itchiness depends, but the fact of its occasionally preceding the jaundice, renders it probable that it is not caused by the bile-pigment. It is rarely observed, except in cases where the jaundice is due to obstruction of the bile-duct.

6. *Cutaneous Eruptions*.—Urticaria and lichen, and sometimes boils and carbuncles, are occasionally observed in connection with jaundice. Dr. Graves* refers to eight or nine cases where persons suffering from acute rheumatism became suddenly jaundiced, from the supervention of ‘hepatitis’ (congestion of liver?) and where the jaundice was followed by urticaria. I have not myself observed this sequence of diseases, but a patient now under my care (Case LXXVIII.), has been suffering from congestion of the liver with jaundice, which supervened shortly after recovery from an attack of acute rheumatism and pericarditis. Here, however, there has been no urticaria.

I must here call your attention to a very remarkable condition of the skin, named *Vitiligoidea*, which has been observed in some rare cases of jaundice, with enlargement of the liver. The attention of the profession was first directed to the subject by the late Dr. Addison and by Dr. Gull, in a joint paper in the Guy’s Hospital Reports,† and two interesting examples of the disease have lately been exhibited to

* Clinical Lect. on the Practice of Medicine, 2nd ed. vol. i. p. 446.

† Guy’s Hosp. Rep. 2nd Ser. vol. vii. 1851, p. 265.

the Pathological Society, by Dr. Hilton Fagge.* The disease presents itself in two forms, either independently, or in combination. In one (*Vitiligoidea plana*), the skin of the eyelids, of the palms of the hands and of the flexures of the fingers, and the membrane of the gums present opaque white patches, with the surface and edges slightly raised, and contrasting strongly with the surrounding jaundiced (or in the case of the gums, red) surface. These patches are not at all indurated, but their sensibility is increased: on close examination the cuticle over them is found to be healthy, and the appearance seems due to some alteration in the cutis. The other (*Vitiligoidea tuberosa*) consists of scattered tubercles of various sizes, some as large as a pea, together with shining colourless papules. The larger tubercles are tense and shining, and not unlike *Molluscum*; but when punctured they give out nothing but blood, and on microscopic examination they have been found to consist of tough fibrous deposit in the true skin, infiltrated with an opalescent fluid, containing fat-granules. They are of a yellowish colour, mottled with a deepish rose tint, and with small capillary veins, here and there ramifying over them, and they are accompanied by a moderate degree of irritation, so that their apices often appear rubbed and inflamed. They are most numerous on the face and ears, on

* Pathological Transactions, vol. xix. A case is also reported by Dr. Pavy, in the Proceedings of the Roy. Med. and Chir. Soc. June 12, 1866.

the outside and back of the forearms, and especially about the elbows and knees, where they are often confluent. The jaundice in these cases is not intense, but is remarkable for its persistence, often lasting for many years; it is not due to obstruction of the ducts, for the motions contain bile. The enlargement of the liver is usually considerable, its lower edge reaching down to the umbilicus; it is usually accompanied by some tenderness, but its precise nature has not yet been determined by post-mortem examination.*

7. *The Temperature* is not materially altered in cases of simple jaundice.

8. *Slowness of Pulse*.—A common result of non-febrile jaundice, is to retard the action of the heart, and diminish arterial tension. The pulse may fall to 50, 40, or even 20, and sometimes it is also irregular. This slowness of pulse is particularly noticeable when the patient is recumbent; when he stands, the circulation becomes considerably quickened. It is also accelerated when there is pyrexia in addition to the jaundice; but when fever precedes, the pulse usually falls on the supervention of the jaundice. Hence in jaundice, the frequency of the pulse is a less reliable indication of fever than under

* It is worth mentioning, that in two cases at least, a similar eruption has been observed, where there has been no jaundice, but where there has been diabetes, an observation of considerable interest when it is remembered how intimately connected the liver is with the pathology of diabetes. (See Addison and Gull, in *Guy's Hosp. Rep.* 2nd Ser. vol. vii. p. 268; and Bristowe, in *Path. Trans.* vol. xvii. p. 414.)

ordinary circumstances, and we must trust mainly to the temperature. Slowness of the pulse is not an invariable symptom in jaundice, but it has not yet been explained why this condition is present in some cases, and absent in others. The natural explanation would be that it is due to one particular ingredient of the bile, which does not exist in the blood in all cases of jaundice. Now some recent experiments of Röhrig upon animals have shown that the biliary acid salts exercise a specific paralysing action upon the heart and retard its action, while bile-pigment has no such effect.* Slowness of the pulse, therefore, in jaundice may indicate the presence in the blood of unchanged biliary acids.

9. *Hæmorrhages*.—In all cases where jaundice lasts a long time, the blood becomes impoverished by a diminution in the proportion of red corpuscles and fibrine, and as a result of this there is sometimes developed a tendency to hæmorrhages from the various mucous membranes. In cases of protracted jaundice from mechanical obstruction, the immediate cause of death is not unfrequently copious hæmorrhage from the stomach or bowels. This tendency to hæmorrhage, it is true, is particularly observed in conjunction with cerebral symptoms and other indications of blood-poisoning, in cases of jaundice where there is no obstruction of the bile-duct, but it also occurs in cases of mechanical jaundice of long standing, when the secreting tissue

* Archiv. für Heilk. Aug. 1863, p. 385.

of the organ has in a great measure disappeared. I have already (page 231) called your attention to the frequency of hæmorrhages in cases of acute atrophy of the liver, where there is no impediment to the flow of bile into the bowels.

10. *General Debility*.—The impaired nutrition and impoverished blood usually induce a condition of general debility and exhaustion, associated with hypochondriasis and irritability of the temper.

11. *Xanthopsy, or Yellow Vision*.—In rare cases of jaundice, all white objects appear to the patient yellow. There is some difference of opinion as to the mode of production of this yellow vision. In several instances, where it was present, Sir Thomas Watson noted a distended condition of the vessels of the conjunctivæ, and he refers to a case of Dr. Elliotson's, where yellow vision was limited to one eye covered with varicose vessels; he accordingly concluded that it was only when the vessels of the eye were large enough to transmit blood-globules, that they give passage to the bile-pigment, which tinges the humours of the eye.* You will remember that there is considerable enlargement of the conjunctival vessels, in the case of William M—— (Case LXIV.), who for a short time had yellow vision, but that although he no longer sees yellow, the enlargement of the vessels remains. The fact that the yellow vision often intermits without any change in the jaundice, and that it is frequently absent when there

* Lect. on the Principles and Practice of Physic, 3rd ed. vol. ii. p. 255.

is intense jaundice of the cornea and of the other tissues of the eye, and the statement that it has been met with in typhus fever where there has been no jaundice, and that it is sometimes associated with other derangements of vision, such as night-blindness, have led many to regard it as a purely nervous symptom. The ophthalmoscope may possibly clear up the existing doubts respecting this curious symptom. It may be mentioned, however, that after the use of santonine yellow vision is observed, which ceases as soon as the colouring matter is eliminated from the blood by the kidneys.

12. *Cerebral Symptoms*, such as acute delirium, stupor, coma, convulsions, muscular tremors, subsultus, carphology, paralysis of the sphincters, a dry brown tongue, and other indications of the 'typhoid state,' occasionally supervene in cases of jaundice. They are most common in cases where there is no obstruction of the ducts, but they may also occur in cases of long-standing obstruction, where all or a greater part of the secreting tissue is destroyed. Different opinions are held as to their cause. After death no lesion of the brain or of its membranes is found to account for them, and they must therefore be due to some alteration in the nature of the blood. They are commonly attributed to poisoning of the blood with bile, and many experiments have been performed on animals to show that bile is a deadly poison. That dogs should die after injection into the cellular tissue of the bile of other dogs is not extraordinary, and admits

of another explanation than that of the essential elements of bile being a poison. The injection of mucus from another dog would probably produce a like result, and all bile contains mucus.

Pure bile, from which the mucus has been removed, has been repeatedly injected into the large veins of dogs, by Frerichs and other observers, without any cerebral symptoms or bad results ensuing, except that death in some instances was caused by the entrance of air into the veins.* The operation has been even repeatedly performed on the same animal without any lasting injury. But it is scarcely necessary to turn to experimental enquiries on the lower animals for any evidence on the matter, and in all these experiments there are many sources of fallacy. You have had abundant proof in the wards, that the blood of the human subject may be saturated with bile for months (and I may add that it may for years) without any cerebral symptoms resulting. Those of you who have witnessed the case of William M—— (Case LXIV.), who for many months has had permanent closure of the bile-duct, will find it difficult to believe that bile is a deadly poison.

A distinguished American physician, Dr. Austin Flint, has recently endeavoured to show that the cerebral symptoms in jaundice are due to the retention of cholesterine in the blood, or to what he has designated *cholestearcæmia*. Cholesterine is a crystalline fatty matter, and is one of the constituents of the

* Diseases of Liver, Syd. Soc. Ed. vol. i. p. 395.

complex substance, bile. Dr. Flint regards it as an excrementitious product of nervous tissue, the elimination of which from the body is one of the functions of the liver.* Arrived in the bowel, the cholesterine, according to him, is converted into *stercorine*, and therefore it is not found in the fæces, but when retained in the blood he believes it to be a poison like urea. But if the non-excretion of all the elements of bile does not give rise to cerebral symptoms, it is difficult to understand how these symptoms can result from the retention of cholesterine alone. In cases, for instance, of permanent closure of the duct, cholesterine is not discharged from the liver into the bowel, nor does it accumulate in the biliary passages, or produce cerebral symptoms if it be retained in the blood. There are moreover many cases on record where there has been permanent closure of the bile-duct, followed by entire destruction of the secreting tissue of the liver, and where in consequence this organ has been incapable of eliminating any of the elements of the bile which may be preformed in the blood, and yet where no cerebral symptoms have been noticed. Arguing from such cases Dr. Budd contends that when cerebral symptoms occur in jaundice, they are due to some peculiarly noxious matter which is evolved, in *consequence of decomposition*, in the lobular substance of the liver.† No such noxious matter, however, has yet been discovered.

* American Journ. of Med. Science, Oct. 1862.

† Dis. of Liver, 3rd edit. pp. 270, 475.

The cerebral symptoms in jaundice certainly resemble those produced by many known blood-poisons, but the poison is more probably generated in the blood, and throughout the body generally, than in the liver in particular. The liver is not merely an excretory organ, but unquestionably exercises an important influence on the metamorphoses of matter constantly taking place in the blood and tissues, although the precise nature of the changes which it effects is but little known. The arrest of the functions of the liver, as for instance in cases of acute atrophy (see p. 224), checks or modifies these metamorphoses. One result is that urea is not elaborated, but substances such as leucine and tyrosine, of a composition intermediate between it and the proteine compound (see page 230), are developed, and the materials which ought to be eliminated from the body as urea and uric acid accumulate in the blood. In acute yellow atrophy, and in the yellow fever of the tropics, the occurrence of cerebral symptoms is marked by an extraordinary diminution of urea in the urine. The pathology in fact of the cerebral symptoms in jaundice is probably identical with what I have endeavoured to prove to you is the pathology of the typhoid state in all diseases.* But to this subject we shall return after considering the theory of jaundice.

* See Abstract of a Clinical Lecture on the Pathology and Treatment of the Typhoid State in different Diseases. Brit. Med. Journ. Jan. 4, 1868.

THEORY OF JAUNDICE.

All cases of jaundice may be referred to one of two classes, viz.—

I. Cases in which there is a mechanical impediment to the flow of bile into the duodenum, and where the bile is in consequence retained in the biliary passages, and thence absorbed into the blood.

II. Cases in which there is no impediment to the escape of bile from the liver.

These two forms of jaundice have long been recognized; but great differences of opinion have been held, and still exist, as to the mode of production of the jaundice in the second class of cases, and yet these are the cases which are, perhaps, the most common in practice.

When any obstruction exists to the flow of the bile through the hepatic or common duct, the way in which jaundice arises is sufficiently clear. The bile-ducts and the gall-bladder become distended with bile, which is absorbed into the blood by the lymphatics and the veins. This was satisfactorily proved at the beginning of this century, by the experiments of Dr. Saunders,* which have since been confirmed by many other observers. If the hepatic duct of a dog be ligatured, and the animal killed after two hours, the lymphatics in the walls of the bile-ducts, which are very numerous, are seen to be distended with a

* Treatise on the Structure, Economy, and Diseases of the Liver, and on Bile and Biliary Concretion, 3rd ed. 1803.

yellow fluid. The fluid in the thoracic duct is also yellow, and so likewise are the intervening lymphatic glands. In patients also who die of obstruction of the bile-duct, the lymphatics of the liver are often found to contain bile. On the other hand, after ligature of the common duct, the serum of blood taken from the hepatic vein two hours afterwards is found to contain much more bile-pigment than that of blood taken from the jugular vein in the neck. This preponderance of bile-pigment in the blood of the hepatic veins over that of the general circulation shows that bile in cases of obstruction of the gall-duct, is also directly absorbed by the veins. Indeed, as we shall presently find, there is reason to believe that even when there is no obstruction, bile is constantly passing from the gall-bladder and biliary passages into the circulation, in virtue of the law of diffusion of fluids through animal membranes. Under ordinary circumstances jaundice does not result, because the bile is at once transformed in the blood, and in its turn influences the metamorphosis of other matters, the products of which metamorphoses are eliminated by the urine. But in the distention of the biliary passages consequent on obstruction, the pressure upon, and the extent of, the diffusing surface are increased, and consequently more bile enters the blood than can undergo the metamorphosis necessary for its elimination by the urine. Even in obstruction, however, the intensity of the jaundice (or the amount of unchanged bile accumulated in the blood) will

vary with the amount of bile secreted by the liver and the activity of oxydation going on in the blood.

But in a large proportion of cases there is no mechanical impediment to the escape of bile from the liver, and then an explanation of the jaundice is less obvious. Boerhaave and Morgagni long ago suggested that the jaundice in these cases was the result of a *suspended secretion*. They maintained that the function of the liver was merely to separate the elements of bile which were already formed in the blood, and that when anything occurred to interfere with the function of the liver, the blood retained the ingredients of the bile, and the result was jaundice of the skin and other tissues. Although this view was strenuously opposed in this country at the beginning of the century by Dr. Saunders, who contended that 'in every case of jaundice, bile must be secreted and carried into the blood vessels,'* it is the view which is generally received at the present day. Dr. Budd, for instance, in his valuable treatise on Diseases of the Liver, remarks, 'in these cases the most obvious explanation of the facts is, that the biliary pigment exists in the blood, and that in consequence of defective action of the secreting cells, it is not eliminated as it should be in the liver.'† It is but right to add,‡ however, that

* Op. cit. p. 107.

† Op. cit. p. 468.

‡ Bile is a very complex substance. Its composition, according to Gorup-Besanez, is as follows:—

Water	822·7 to 908·1
Solid matter	177·3 „ 91·3
Bile-acid salts	107·9 „ 56·5
Fat and cholesterine	47·3 „ 30·9
Mucus and pigment	23·9 „ 14·5
Ash	10·8 „ 6·3

Dr. Budd makes a special exception with regard to the biliary acids. 'The most skilful chemists,' he says, 'who have recently analysed the portal blood, have failed to detect the biliary acids in it, and have come to the conclusion that these at least, are formed in the liver.'* This view, that the liver manufactures the bile-acids, while it merely excretes the bile-pigment, is also the one adopted by Dr. G. Harley, in his essay on Jaundice.†

It seems to me, however, that there are weighty objections to the view, that even the bile-pigment is formed in the blood, and merely excreted by the liver, some of which may be mentioned.

1. Although it is very probable, that bile-pigment is derived from the colouring matter of the blood, and may be produced from this by the action of chemical reagents, or may even be developed in extravasations as a pathological product,‡ it has not

Two acids have been found in the bile which have been named by Lehmann, glycocholic and taurocholic acid. According to this chemist these acids are formed by the conjugation of cholic acid with glycine (gelatine-sugar), and taurine respectively, and they are united in the bile with soda as a base. Lehmann makes the composition of glycocholic acid, $C_{52}H_{42}NO_{11}HO$, and that of taurocholic acid, $C_{52}H_{45}NS_2O_{14}$. Two modifications of bile-pigment have been found, viz. a brown pigment, named cholepyrrhine, bilifulvine, or bilirubine, and a green pigment, biliverdine; cholepyrrhine is convertible into biliverdine. Very little is known of the chemical nature of these substances; there are probably other modifications of the pigmentary matter, which, as well as those mentioned, are the products of the transformation or oxydation of one primitive substance.

* Op. cit. pp. 40, 467.

† Jaundice, its Pathology and Treatment, by G. Harley, M.D. Lond. 1863.

‡ See Virchow's Cellular Pathology, Engl. Transl. pp. 128, 145, and Kuhne, Lehrbuch der physiologischen Chemie, Leipzig, 1866, p. 89.

yet been satisfactorily shown that bile-pigment exists ready formed in the blood of persons who have not jaundice. Frerichs, no mean authority, denies that it ever has. Lehmann, who has investigated with great care the changes which the blood undergoes in passing through the liver, has never been able to detect the colouring matter of bile in portal blood, and infers that this as well as the bile-acids must be formed in the liver itself.* It is obvious then that the quantity of bile in healthy blood must be at all events very minute; and when it is remembered that, according to Bidder and Schmidt, the daily quantity of bile manufactured in the liver is about 56 ounces avoird., and yet that jaundice is not a normal condition, it seems equally clear that the bile-pigment must be formed in the liver. It is not probable that part is formed in the blood, and part by the liver.

2. The discovery occasionally of a small quantity of bile-pigment in normal blood would not prove that it was formed in the blood, for it is quite conceivable that it may have been formed in the liver and been then absorbed. It is probable, indeed, as I shall endeavour to prove to you presently, that bile-pigment is constantly being absorbed into the blood, becoming altered in the act of absorption or immediately after; and if this be so, it is quite possible that a trace of it

Analyses of the bile-pigments, which have been recently communicated to the Royal Society by Dr. Thudichum, tend to show that they have no relation to hæmatine, as was formerly supposed. (Proc. Roy. Soc. 1867, vol. xvi. p. 220.)

* Physiological Chemistry, Dr. Day's transl. vol. ii. p. 87.

should occasionally remain unaltered without giving rise to obvious jaundice.

3. If bile-pigment were ready formed in the blood, the jaundice in those cases where the secretion is suppressed ought to be quite as intense as that which is produced by an obstruction of the bile-duct (or more so), but the reverse is the case.

4. If bile-pigment were ready formed in the blood, intense jaundice ought to follow at once the extirpation of the liver in any of the lower animals. So far from this, Müller, Kunde, Lehmann, and Moleschott have repeatedly extirpated the liver of frogs, and have invariably failed to find a trace either of the biliary acids or of the colouring matter of the bile in the blood, the urine, or the muscular tissue.*

5. It not uncommonly happens that in consequence of various diseases, such as fatty and waxy degeneration, cancer and cirrhosis, the secreting tissue of the liver in the human subject is for the most part or entirely destroyed, bile is no longer secreted, and yet no jaundice results. Several cases of this sort are referred to by Haspel, where the gall-bladder after death contained only a little white mucus.† Frerichs also records a case of fatty liver where the contents of the bowels were pale, the gall-bladder empty, and the biliary ducts coated with a greyish mucus, notwithstanding which the skin was of a chalky paleness, and the urine contained no bile-pigment.‡ Similar

* Carpenter's Physiology, 5th ed. p. 373.

† Malad. d'Algérie, i. 262.

‡ Op. cit. Eng. Ed. i. 83.

observations have been made by Dr. Budd, in cases of waxy disease and cancer of the liver,* and several instances of a like nature came under my notice while I was pathologist to the hospital. If bile-pigment were formed in the blood, it would be difficult to account for what becomes of it in these cases.

These considerations make it very doubtful if any form of jaundice can with propriety be attributed to a suppression of the hepatic functions. It remains then to be considered if any more satisfactory explanation can be offered of those cases of jaundice in which there is no impediment to the flow of bile from the liver into the duodenum.

A solution of the difficulty has been proposed by Professor Frerichs, of Berlin. According to this distinguished observer, a large proportion of the colourless biliary acids found in the liver is either directly taken up by the blood in the hepatic vein, or is absorbed from the bowel. It is believed that under ordinary circumstances, the biliary acids undergo metamorphosis from oxydation, and assist perhaps in forming the large quantity of taurine found in healthy lung and the pigments which are voided in the urine, but that these natural transformations are liable to interruption by nervous agencies, or by poisons in the blood, and that then the bile-acids, not being sufficiently oxydized, are converted into bile-pigment.† This view has been supported by two experiments intended to show: 1. That bile-pigment can be obtained artificially from the bile-acids by the

* *Op. cit.* pp. 329, 411.

† *Op. cit.* vol. i. pp. 89, 394.

action of concentrated sulphuric acid; and, 2. That colourless biliary acids injected into the veins of dogs are converted in the blood of these animals into bile-pigment. These alleged facts, as well as the conclusions drawn from them, are still the subject of much discussion. They have been controverted by Kulne* Hoppe, Harley,† &c., but supported by Staedeler,‡ Neukomm,‡ Folwarczny,‡ and Röhrig,§ &c. It is contended on the one hand, that the biliary acids are decomposed in the blood, and on the other that in whatever manner they find their way into the blood, they are excreted unchanged by the kidneys. Although the majority of observers seem to concur with Frerichs, it would be waste of time to endeavour to extract the truth from such conflicting statements. Nor is the decision of the question at issue perhaps of material importance, for explaining those cases of jaundice in which there is no impediment to the escape of bile from the liver.

There are grounds for believing that not only in jaundice, but in health, a portion of the bile-pigment, as well as of the bile-acids formed in the liver, is absorbed into the blood.||

* Virchow's Archives, vol. xiv. pts. 3 and 4, Sept. 1858, and Beale's Archives of Medicine, vol. i. p. 342.

† Pathology and Treatment of Jaundice, 1863.

‡ See Translator's Preface to the English Edition of Frerichs on the Liver, pp. xv., xvi.

§ Archiv. für Heilkunde, Aug. 1863, p. 385.

|| It may be thought improbable that the liver should secrete from the portal vein a material which is afterwards to be absorbed by the branches of the same vessel. But it has been too readily assumed from the comparatively large size of the vena portæ that it furnishes

1. The quantity of bile-pigment discharged with the *fæces* is but a fraction of what is calculated to be secreted by the liver. Speaking of the principal constituents of bile, Dr. Carpenter* remarks : ' the further we descend in the intestinal canal, the less of them do we meet with : ' and again he says : ' of the bile which is poured into the alimentary canal, a large part is certainly re-absorbed, its constituents being all the materials of bile. It is probable that the portal vein ministers chiefly to the assimilating function of the liver, and the hepatic artery to that of secretion, or, in other words, that the special function of the portal vein is to convey from the digestive canal to the liver the carbonaceous materials of the food (fat, amylacea, sugar, and alcohol), to be there manufactured into a glycogenic material, which in its turn is converted into fat, and that the biliary acids and bile-pigment are secreted from arterial blood, like the urea and uric acid in the kidney. At all events, there are facts which show that bile may be formed quite independently of the portal vein.

1. In the ' *Philosophical Transactions* ' for 1793, a case is recorded where the portal vein passed direct to the *vena cava inferior*, without entering the liver, and yet where bile was found both in the intestines and gall-bladder. Other cases of a similar nature are referred to by Dr. Carpenter in his ' *Principles of Human Physiology*, ' 5th ed. p. 372.

2. There are many cases on record where there has been complete obstruction of the portal vein from disease, for some time before death, and yet where bile has continued to be secreted. (See for instance, *Frerichs*, *Op. cit.* i. 274, and *Gintrac*, *L'oblitération de la veine porte*, Bordeaux, 1856 ; also *Dickinson*, *Path. Trans.* vol. xiv. p. 63.)

3. In animals with a portal vein this vessel has been tied, and yet bile has been secreted ; and although the quantity has been reduced, this may have been due to the want of those substances (fat, &c.), ordinarily supplied by the portal vein. It is not probable that the same materials should be secreted both by the portal vein and the hepatic artery (*Carpenter's Prin. of Human Physiology*, p. 372, and *Comparative Physiology*, p. 424).

4. In the *Invertebrata* the liver is supplied with blood from an arterial source only (*Carpenter's Prin. of Comparative Phys.* 4th ed. p. 422).

* *Carpenter's Principles of Human Physiology*, 5th ed. pp. 102, 353, 374.

destined to undergo oxydation and be eliminated, for the most part by the respiratory processes : and it is probably from this reabsorbed portion of the bile that the sulphur of the urine is derived.* According to Dr. Bence Jones, also, ‘the colouring matter (of the bile) undergoes changes in the intestines, and some of it most probably in health is carried into the blood and textures, and is finally removed in the colouring matter of the urine.* It is the knowledge of this circumstance that offers the only satisfactory explanation of the remarkable discrepancy of opinion in the profession respecting mercury, podophyllin, and other substances which are supposed to exercise some specific effect upon the liver, in stimulating it to an increased secretion of bile. The practical physician gives a dose of calomel, finds the quantity of bile in the motions greatly increased, and argues that the liver has been stimulated to an increased secretion ; but the physiologist ties the common bile-duct, makes a fistulous opening into the gall-bladder, and then finds that calomel has no effect on, or even diminishes, the amount of bile that drains away through the fistula.† Mercury and allied purgatives probably produce bilious stools by irritating the upper part of the bowel, and sweeping on the bile before there is time for its absorption ; irritating articles of diet will often produce precisely the same effect. Calomel is of

* St George’s Hospital Reports, vol. i. p. 192.

† On the Influence of Mercurial Preparations on the Secretion of Bile, by George Scott, M.D. Beale’s Archives of Medicine, vol. i. 209.

unquestioned utility in congestion of liver, but if it acted, as is usually argued, by stimulating the liver to increased secretion, it might be expected to increase the congestion rather than diminish it. It is quite possible, however, that the irritation of the duodenum by purgatives may be reflected to the gall-bladder, and cause it to contract, and that the evacuation of this viscus may account in part for the increased quantity of bile in the stools.

2. Although in the human subject, if there be no obstruction of the common bile-duct, sufficient pigment remains to colour the *fæces*, in carnivorous animals the *fæces* contain scarcely any pigment, and in the *boa*, although the liver is large and secretes bile freely, the excrement contains no trace of pigment. The bile-pigment in these animals disappears for the most part or entirely in the bowel, and yet these animals are not jaundiced.

3. From what is now known of the diffusibility of fluids through animal membranes, it is impossible to conceive bile long in contact with the lining membrane of the gall-bladder, bile-ducts, and intestine, without a portion of it (including the dissolved pigment) passing into the blood. A circulation is constantly taking place between the fluid contents of the bowel and the blood, the existence of which, till within the last few years, was quite unknown, and which even now is too little heeded.* ‘It is now

* For instance, the purging of cholera is probably the result of some stoppage in this intestinal circulation—of a diminished power of absorb-

known,' says Dr. Parkes, in his *Gulstonian Lectures on Pyrexia*, 'that in varying degrees, there is a constant transit of fluid from the blood into the alimentary canal, and as rapid reabsorption. The amount thus poured out and absorbed in twenty-four hours is almost incredible, and of itself constitutes a secondary or intermediate circulation never dreamt of by Harvey. The amount of gastric juice alone, passing into the stomach in a day, and then reabsorbed, amounted in the case lately examined by Grunewald,* to nearly 23 imperial pints. If we put it at 12 pints we shall certainly be within the mark. The pancreas, according to Kroeger, furnishes $12\frac{1}{2}$ pints in twenty-four hours, while the salivary glands pour out at least 3 pints in the same time. The amount of the bile is probably over 2 pints. The amount given out by the intestinal mucous membrane cannot be guessed at, but must be enormous. Altogether the amount of fluid effused into the alimentary canal in twenty-four hours amounts to much more than the whole amount of blood in the body; in other words, every portion of the blood may, and possibly does, pass several times into the alimentary canal in twenty-four hours. The effect of this continual outpouring is supposed to be to aid metamorphosis; the same substance more or less changed seems to be thrown out and reabsorbed

tion, rather than of an increased exhalation from the mucous membrane of the bowel. Numerous facts render it probable that in cholera the power of absorption is greatly impaired or abolished.

* An account of this case, abstracted by me from Grunewald's Latin Memoir, will be found in Beale's *Archives of Medicine*, vol. i. p. 270. C.M.

until it be adapted for the repair of tissue or become effete.' *

It is in the course of this osmotic circulation that the constituents of bile are taken up into the blood, becoming themselves probably transformed in the process into products which are eliminated by the lungs and kidneys,† while at the same time they assist in the assimilation of the nutritive materials derived from the food. And here we have an explanation of those cases of jaundice where there is no impediment to the flow of bile from the liver. Under normal conditions, the bile that is absorbed is at once transformed, so that neither bile-acids nor bile-pigment can be discovered in the blood, and there is no jaundice. But in certain morbid states the absorbed bile does not undergo the normal metamorphoses, but circulates with the blood and stains the skin and other tissues. The morbid states, which, so far as we know, conduce mainly to this result, are these :—

1. Certain poisons, such as snake-poison and chloroform, the poisons of yellow fever, relapsing fever, and pyæmia, or in rarer cases, those of remittent fever, typhus, scarlatina, &c.

2. Nervous influences, such as a sudden fright, great or protracted anxiety, &c.

3. A deficient supply of oxygen, as in persons living

* Med. Times and Gazette, April 7, 1855, p. 333.

† In various diseased conditions of the liver, even when there is no jaundice, or bile-pigment in the urine, this fluid is rendered very dark, sometimes almost black, by boiling and adding nitric acid.

in confined and crowded dwellings, may prevent the normal metamorphoses of bile taking place.

4. An excessive secretion of bile, especially when conjoined with constipation. In this case, unless the bile be removed by the bowels, too much may be absorbed to undergo the normal metamorphoses, and the presence of the untransformed bile in the blood causes jaundice.

The only difference then between jaundice from obstruction, and jaundice independent of obstruction of the common bile-duct, is that in the former case none of the bile can escape from the body by the fæces, and consequently all that is secreted, after the gall-bladder and biliary passages are fully distended, must be absorbed into the blood. As might be expected, the jaundice in this form is usually much more intense than in the other, although even here the intensity will vary according to the amount of bile secreted by the liver, and the activity of the metamorphoses going on in the blood. When the obstruction has lasted long, the jaundice often becomes much paler, not from removal of the obstruction, but from the secreting tissue of the liver being destroyed and comparatively little bile being secreted.

Lastly, we may enquire what explanation the theory of jaundice now advanced gives of the cerebral symptoms met with in certain cases and already referred to (p. 296). From what has been stated it is very probable that the entrance of bile into the blood is necessary to perfect those metamorphoses from which

materials for the urinary solids are derived. At all events, this seems certain that when the secreting tissue of the liver is destroyed, as in acute atrophy and in certain cases of long-standing obstruction of the bile-duct, these metamorphoses are imperfectly executed. Urea is not formed in sufficient quantity, and substances such as leucine and tyrosine, of intermediate composition between it and the proteine compounds (see p. 230), accumulate in the blood and appear in the urine. These are the circumstances under which cerebral symptoms occur in cases of so-called 'suppression of bile.' The mere presence of bile in the blood, as I have already shown you (p. 297), will not account for them, and indeed in those cases where cerebral symptoms are most apt to supervene, the jaundice as a rule is less intense than it often is when they are absent.

The detailed consideration of the various causes of jaundice, and of the means of distinguishing them, we must reserve for subsequent lectures.

LECTURE IX.

*JAUNDICE.*CLASSIFICATION OF CAUSES OF JAUNDICE—JAUNDICE
FROM OBSTRUCTION OF THE BILE-DUCT.

GENTLEMEN,—After the preliminary remarks on the subject of jaundice made in a former lecture, we may now proceed to consider its different causes, and the means of distinguishing them.

All cases of jaundice, as I have told you, may be conveniently grouped under the two heads of—

A. Jaundice resulting from Obstruction of the Common Bile-Duct; and

B. Jaundice independent of any obstruction of the Bile-Duct.

The numerous causes comprised under each of these heads may be seen from this Table:

TABULAR VIEW OF THE

A. JAUNDICE FROM MECHANICAL OBSTRUCTION OF THE BILE-DUCT.

I. OBSTRUCTION BY FOREIGN BODIES WITHIN THE DUCT.

1. Gall-stones and inspissated bile.
2. Hydatids and Distomata.
3. Foreign bodies from the intestines.

II. OBSTRUCTION BY INFLAMMATORY TUMEFACATION OF THE DUODENUM, OR OF THE LINING MEMBRANE OF THE DUCT WITH EXUDATION INTO ITS INTERIOR.

III. OBSTRUCTION BY STRICTURE OR OBLITERATION OF THE DUCT.

1. Congenital deficiency of the duct.
2. Stricture from perihepatitis.
3. Closure of orifice of duct in consequence of an ulcer in the duodenum.
4. Stricture from cicatrization of ulcers in the bile-ducts.
5. Spasmodic stricture?

IV. OBSTRUCTION BY TUMOURS CLOSING THE ORIFICE OF THE DUCT OR GROWING IN ITS INTERIOR.

V. OBSTRUCTION BY PRESSURE ON THE DUCT FROM WITHOUT, BY

1. Tumours projecting from the liver itself.
2. Enlarged glands in the fissure of the liver.
3. Tumour of the stomach.
4. Tumour of the pancreas.
5. Tumour of the kidney.
6. Postperitoneal or omental tumour.
7. An abdominal aneurism.
8. Accumulation of fæces in bowels.
9. A pregnant uterus.
10. Ovarian and uterine tumours.

CAUSES OF JAUNDICE.

B. JAUNDICE INDEPENDENT OF MECHANICAL OBSTRUCTION OF THE BILE-DUCT.

I. POISONS IN THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

1. The Poisons of the various specific fevers :
 - a.* Yellow fever.—*b.* Remittent and Intermittent fevers.—*c.* Relapsing fever.—*d.* Typhus.—*e.* Enteric or Pythogenic fever.—*f.* Scarlatina.—*g.* Epidemic Jaundice.
2. Animal Poisons :
 - a.* Pyæmia.—*b.* Snake-poison.
3. Mineral Poisons :
 - a.* Phosphorus.—*b.* Mercury.—*c.* Copper.—*d.* Antimony, &c.
4. Chloroform and Ether.
5. Acute Atrophy of the liver?

II. IMPAIRED OR DERANGED INNERVATION INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

1. Severe mental emotions, fright, anxiety, &c.
2. Concussion of the brain.

III. DEFICIENT OXYGENATION OF THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

IV. EXCESSIVE SECRETION OF BILE, MORE OF WHICH IS ABSORBED THAN CAN UNDERGO THE NORMAL METAMORPHOSIS.

Congestion of the Liver :

- a.* Mechanical.—*b.* Active.—*c.* Passive.

V. UNDUE ABSORPTION OF BILE INTO THE BLOOD FROM HABITUAL OR PROTRACTED CONSTIPATION.

I shall now endeavour to describe to you the distinguishing characters of the several forms of jaundice referred to in the Table.

A. JAUNDICE FROM MECHANICAL OBSTRUCTION OF THE BILE-DUCT.

I. OBSTRUCTION BY FOREIGN BODIES WITHIN THE DUCT.

1. *Gall-stones or Inspissated Bile.*

Gall-stones are among the most common causes of Jaundice from Obstruction. It very commonly happens that the gall-bladder is found full of concretions after death, as in the specimens I show you here, and yet that there have been no symptoms during life to lead to any suspicion of their existence. Gall-stones only produce jaundice and other symptoms when they enter the bile-duct, and the most characteristic symptoms are those which are produced by the *passage of the concretions along the duct*. In most cases where there are the symptoms of gall-stones, there is a distinct concretion or calculus; but similar symptoms occasionally result from what is called inspissated bile or a gritty condition of the bile. It is not often that you have an opportunity of proving this by post-mortem examination, although it is a fact of some clinical importance, inasmuch as it accounts for some of those cases where there have been the symptoms of gall-stones, but where none

can be found in the stools. You will find, however, a case related by Dr. Handfield Jones, in the fifth volume of the 'Pathological Transactions' (p. 150), where a woman died of universal jaundice, a short time after having fractured her thigh by a fall, and where the lower end of the common duct was found quite plugged up with 'a sandy matter consisting of biliary pigment.'

The jaundice resulting from gall-stones as a rule is not difficult to diagnose.

1. The passage of a gall-stone along the common duct, unless it be a very small one, gives rise to the pain known as biliary colic. This pain generally comes on suddenly and unexpectedly, shortly after a meal, or after some severe muscular exertion. The pain is referred to the gall-bladder, and extends from this round to the right scapula, to the neck, or to the back. It usually is of two sorts, a dull aching pain which is constant, and an acute agonizing pain which comes and goes in paroxysms, and which is described as of a boring, tearing, burning, or constricting character. The latter is often so excruciating, that the patient will bend himself double, roll about the floor, press his hands against his stomach; and in nervous persons it may even excite epileptiform convulsions. These paroxysms induce great exhaustion, the face being pale, the pulse slow, and the whole body covered with a cold sweat; occasionally this condition passes into syncope, which in rare cases has been known to be fatal. With this pain there is not

necessarily any tenderness, and the pain may even be relieved by pressure, but after it has lasted long there is usually some tenderness over the fundus of the gall-bladder, and occasionally this tenderness is acute, from the lining membrane of the gall-bladder and bile-ducts having become inflamed.

2. Rigors, often severe, recurring at irregular intervals, but sometimes periodically almost with the exactness of an ague, are not uncommon in severe and protracted cases, and are believed to depend on over-distention of the gall-bladder and bile-ducts. In reference to this symptom a remarkable specimen is preserved in the Pathological Series of the Royal College of Surgeons.* It is that of a large oval calculus fitted tightly into the end of the common bile-duct, a portion of it projecting through the dilated orifice of the duct into the duodenum. The patient, from whose body the preparation was obtained, was a very large woman, aged 70, who for nearly six months had been subject to spasmodic pains of the stomach, coming on with shiverings like an ague-fit, which lasted for half an hour or an hour and were succeeded by unusual heat. It was only during the last month of life that vomiting and jaundice had set in. Three days before death she was seized with an unusually severe attack of shivering and pain, which continued, with scarcely any remission, until death.

* The preparation is from the Collection of Mr. John Howship, and is numbered 1459.

3. Vomiting accompanies the pain in most cases and is often frequent and severe, the patient rejecting all food that may be in the stomach and bringing up large quantities of acid fluid. Sometimes also there is frequent hiccup.

4. After these symptoms have lasted for a day or two jaundice usually appears, and if the jaundice continue for a few days it usually becomes intense, the urine being loaded with bile-pigment and the fæces containing none. If the stone, however, find its way quickly into the bowel it is quite possible to have biliary colic without jaundice, and sometimes the urine contains bile-pigment without jaundice ever showing itself in the skin. The duration of the jaundice will vary with the number and size of the stones, but usually it does not last longer than from a few days to a few weeks. It is not very often that a gall-stone leads to permanent jaundice, for if it succeed in escaping from the cystic duct, where its presence will not cause jaundice, it will usually find its way through the larger common duct. Cases, however, are occasionally met with where permanent and even fatal jaundice has been caused by the impaction of a gall-stone.* But although the jaundice of gall-stones be in most cases of temporary duration, it has

* In the 'Pathological Transactions,' a case of fatal jaundice is recorded by Dr. Handfield Jones (vol. v. p. 146), where the hepatic and common ducts of the liver were obstructed by large calculi; and another is reported by Dr. J. Wale Hicks, where the cystic duct and part of the common duct were occupied by a large gall-stone, which also projected into the gall-bladder (vol. xv. p. 126).

this peculiarity that it is liable to recur with the other symptoms already referred to at irregular intervals, owing to some of the concretions not escaping from the gall-bladder during the first attack, or to fresh ones forming in the place of those which have been discharged. The diagnosis then is often materially assisted by the patient having had a similar attack on some former occasion. The very fact of a person in middle or advanced life having had several attacks of well-marked jaundice, with distinct intermissions, would point to gall-stones as the probable cause. At the same time you must remember that when a large calculus has forced its way through the natural channels of the bile, they will remain permanently dilated, and smaller stones may be afterwards voided without either jaundice or pain. According to Sir Thomas Watson, there are persons who get rid of scores of stones in this way, during the course of their lives.

5. The jaundice from gall-stones is unaccompanied by fever; there is no increase of temperature or acceleration of the pulse. There may however be a quick pulse during the paroxysms of pain in nervous persons: and if the pressure or passage of the calculus have induced inflammation of the biliary passages, not only may the circulation be accelerated but the temperature may be elevated.

6. If the jaundice be of some standing there will often be found a slight and uniform enlargement of the liver, with a pyriform tumour corresponding to the gall-bladder, resulting from the great dilatation

of the biliary passages by the accumulated bile, as I have explained to you in a former lecture (p. 142).

7. The sex, age, and habits of the patient are of some use in diagnosis. Gall-stones are much more common in females than in males (3 to 2), and are chiefly met with in persons of middle and advanced life who have led sedentary lives. Of 395 cases collected by Hein, only fifteen were under twenty-five years of age, and only three under twenty. In a previous lecture I have detailed to you a case where gall-stones occurred at the age of twenty-three (Case XXXV. p. 158). There are also rare cases on record where gall-stones have been observed in children under twelve, and in the first volume of the 'Northern Journal of Medicine' (p. 240), you will find a case recorded where fatal jaundice in a new-born babe was due to obstruction of the bile-duct by 'an indurated cord-like plug of inspissated bile.'

8. But the most conclusive proofs of jaundice being due to gall-stones is finding the concretion in the fæces. It is not only satisfactory to see the stones, but their appearance is often of some use in prognosis. If one large globular concretion have been passed, it is very possible that the patient may not be further troubled: but if the stone be marked by several flat surfaces or facets, such as I show you here, the probability is that there are several or many more. But even when all the symptoms above described have been present in a marked degree, you will often fail in finding a gall-stone in the fæces.

This may be due to the concretion becoming disintegrated in the bowel, or to its slipping back into the gall-bladder instead of into the duodenum, or to the obstruction of the duct having been caused by inspissated gritty bile, rather than by a distinct concretion; but too often it is the result of a faulty method of search. The common belief is that gall-stones are lighter than water, and that therefore if water be poured on the fæces, any gall-stones present will float; but Sir Thomas Watson, who recommended this method in the earlier editions of his Lectures, added:—‘I never but once succeeded in thus catching a concretion in the evacuations of a patient, where symptoms had led me to search for it.’ In a later edition, however, he says that three other patients, taught how to search for them, had detected in the alvine discharges this palpable source and explanation of their previous sufferings.* The truth is that most gall-stones, before they are dried, are heavier than water, in which they will not float; and accordingly the plan which you have seen followed in the wards, and which is the only reliable one, is to pass the whole evacuation from the bowels through muslin or a sieve.

2. *Hydatids and Distomata in the Bile-Ducts.*

Hydatid tumours of the liver, as I have already told you (see p. 67), occasionally burst into the bile-duct.

* Lect. on Practice of Physic, 2nd ed., ii. p. 527, and 3rd ed. ii. p. 555.

If the tumour contain no secondary cysts, its fluid contents may be discharged through the bile-duct into the duodenum, and the patient may get well without any marked symptoms arising. But in most cases there are secondary cysts which enter and obstruct the bile-duct, and produce all the symptoms of jaundice from an impediment to the flow of bile. In a former lecture I have related to you two cases where this occurred (Case IX. p. 90, and Case X. p. 94). The passage moreover of the hydatid cysts along the bile-duct may give rise to severe paroxysms of pain, rigors, and vomiting, and in fact to all the phenomena of the biliary colic resulting from gall-stones. This happened, you will remember, in a marked manner in Case X. From gall-stones, however, the case would be distinguished—

1. By there being the physical signs of hydatid enlargement of the liver already described to you (p. 54), with perhaps a subsidence of the swelling on the occurrence of pain.

2. By there being in most cases symptoms of fever, a quick pulse and elevated temperature, in addition to those of biliary colic. When the hydatid bursts into the bile-duct, not only do vesicles enter the duct, but bile enters the hydatid, and the consequence is that this inflames and suppurates and causes fever. Where, however, as in Case X., vesicles continue to pass along the bile-duct long after the bursting of the tumour, there may be biliary colic without fever.

3. The diagnosis will be complete on detecting

hydatid vesicles, in the alvine evacuations, as was done in Case X. (p. 94).

In those rare instances to which I have already (p. 68) directed your attention, where an hydatid tumour appears to be developed in the first instance in the bile-duct, its diagnosis will probably be impossible.

In cases of extreme rarity the *Distoma hepaticum*, or liver-fluke, which is so common in the livers of sheep, has been found in the biliary passages of the human subject, but its presence does not appear necessarily to obstruct the duct and cause jaundice. In Davaine's great work on Entozoa, the case is related of a girl, aged 8, who died in the hospital at Milan of diarrhœa, marasmus, and convulsions, and on opening whose body there was found a pouch containing five distomata near the termination of the common bile-duct. This patient had suffered from all the symptoms of biliary colic, but had no jaundice.* Some years ago a patient died in this hospital whose gall-bladder was found to contain a fluke. The lining membrane of the gall-bladder was perfectly white, but Dr. Budd, who relates the case, does not state whether there was any obstruction of the common duct or jaundice.† The distoma in the sheep causes dilatation and catarrh of the biliary passages, with atrophy of the hepatic tissue and great anæmia, but only in rare cases jaundice. The diagnosis of distomata in the

* *Traité des Entozoaires*, 1860, p. 252.

† *Dis. of Liver*, 3rd ed., p. 494.

bile-ducts of the human subject could only be arrived at in the event of any of the parasites being ejected by vomiting or in the stools.

3. *Foreign Bodies from the Intestine.*

Foreign bodies occasionally find their way from the bowel into the bile-duct, which becomes obstructed, and jaundice is the result. There are not a few instances where round worms have penetrated the orifice of the bile-duct, and caused jaundice, with biliary colic, vomiting, and all the symptoms of gall-stones.* Several of these cases have proved fatal suddenly by convulsions. It is also worth noting that in many of these cases the bowels have contained a number of worms, and that there has been a history of worms being ejected by vomiting, or passed per anum. It is by such an occurrence alone that any diagnosis of the cause of the jaundice could be arrived at.

Foreign bodies, such as cherry-stones and currant-seeds have been known to enter the bile-duct from the intestine, and give rise to jaundice. But in those rare cases where this has happened, it is probable that the bile-duct has been already dilated by the passage of a gall-stone. Several curious cases are on record where the nucleus of a gall-stone has been found to be a dried-up round worm, the fragment of a distoma, a needle, or a plum-stone.

* Frerichs, Dis. of Liver, Eng. Transl., ii. p. 482, and Davaine, Op. cit. p. 156.

II. JAUNDICE FROM OBSTRUCTION BY INFLAMMATORY
TUMEFACTION OF THE DUODENUM, OR OF THE LINING
MEMBRANE OF THE BILE-DUCT WITH EXUDATION
INTO ITS INTERIOR.

When a mucous membrane inflames, it becomes swollen from the increased amount of blood in its vessels and from œdematous infiltration of the submucous tissue, while at the same time the secretion from the surface is increased in quantity and altered in quality. If these changes take place in the mucous membrane lining a narrow tube like the bile-duct, one can easily understand that the passage through it should be blocked up, and this in fact is what often happens. Catarrhal inflammation is one of the most common causes of mechanical jaundice, and is probably the most common cause of jaundice in young persons. Its symptoms and the circumstances under which it occurs have been fully described to you in a former lecture (p. 131). In a large number of cases, as I told you, the inflammation commences in the duodenum, and spreads up the bile-duct, and sometimes the duodenal orifice of the duct may be found effectually blocked up by the tumid mucous membrane of the duodenum or by a plug of viscid mucus, without the inflammation having extended further up the duct.

In diagnosing the causes of jaundice it is important to remember that inflammation of the biliary passages may be caused by gall-stones, and that thus the symptoms of these two causes of jaundice may coexist; or

it is possible that inflammation of the biliary passages may be excited by gall-stones which have never produced biliary colic. In the case of J. K. (Case XXIX. p. 137) you will remember that the inflammation of the biliary passages seemed to be excited by gall-stones, which were found in the gall-bladder and in the bile-ducts, and yet that the most careful enquiry failed to elicit any history of biliary colic. The paroxysmal pain is caused by the passage of the calculus *along the duct*, and particularly through the duodenal opening. Concretions which never leave the gall-bladder may also excite inflammation of its lining membrane which may spread to the bile-ducts, but will not give rise to biliary colic.

III. JAUNDICE FROM OBSTRUCTION TO THE FLOW OF BILE BY STRICTURE OR OBLITERATION OF THE BILE-DUCT.

1. *Congenital Deficiency or Obstruction of the Duct.*

I have already told you that in the majority of cases of the so-called *icterus neonatorum* the yellow colour of the skin is not jaundice at all (p. 282). At the same time infants are liable to real jaundice which is sometimes a serious symptom. It may, as we have seen, depend upon a plugging of the duct with inspissated bile (p. 323), and then there may be some hope of the obstruction giving way and of the child recovering; or, as I shall have occasion to explain to you in another lecture, it may depend on deficient oxygen-

ation of the blood interfering with the normal metamorphosis of bile. At other times it has a pyæmic origin, and is associated with peritonitis, or with phlebitis of the umbilical vein; and lastly it may be due to a congenital closure, obliteration, or absence of the bile-duct, no trace of it remaining, except a little areolar tissue between the hepatic artery and portal vein. The gall-bladder in these cases is extremely small and collapsed, and sometimes it also is absent; but in the duodenum the opening of the pancreatic duct may be found as usual. Cases of this sort have been recorded or collected by Dr. A. D. Campbell, in the 'Northern Journal of Medicine' for 1844, by Dr. Wilks, in the 13th volume of the 'Pathological Transactions' (p. 119), by Dr. West in his standard work on the 'Diseases of Infancy and Childhood' (5th ed. p. 605), and more recently by Dr. Binz, of Bonn, in Virchow's Archives.* In not a few of the cases which have been recorded, there has been evidence of intra-uterine peri-hepatitis, and strange to say, notwithstanding the rarity of the malformation, many writers have referred to several instances of it occurring in the same family. These considerations suggest the desirability of enquiry whether these malformations be not sometimes one of the results of peri-hepatitis from hereditary syphilis.

* Zur Kenntniss des tödtlichen Icterus der Neugeborenen aus Obliteration der Gallengänge. Archiv. f. path. Anat. und Physiol. und f. klinische Medicin, Berlin, 1866, vol. xxxv. p. 360. References to several other papers on the subject will be found in Dr. West's work quoted above.

The following case (Case LX.) came under my own notice a few years ago, in the out-patient department of this hospital.

Jaundice from this cause may be recognized by the following characters:—

a. The jaundice appears within a few days of birth, and gradually increases in intensity. The conjunctivæ as well as the skin are yellow.

b. The motions are white, and the urine leaves a yellow stain on the clothes.

c. In most cases there have been observed hæmorrhages from the umbilicus (often fatal), from the bowels, beneath the skin, and in other parts of the body, as in Case LX.

d. At first the child may appear strong and healthy, but very soon progressive atrophy sets in, attended often with vomiting and diarrhoea, and death usually occurs within a few months of birth. In one of Dr. Campbell's cases and in another related by Dr. West, the infant lived for six months.

2. *Stricture of the Bile-Duct from Peri-hepatitis.*

In peri-hepatitis the lymph which is thrown out becomes after a time organized, and causes thickening of the capsule and firm fibrous bands of adhesion connecting the liver to surrounding parts. Now and then it happens that the new areolar tissue, which is formed in this way in the portal fissure, exerts a constricting effect on the bile-duct, and sometimes also on the portal vein, and the result is, in the one case

jaundice, and in the other ascites with the other signs of portal obstruction already referred to (see p. 241). You will find a case of this sort related by Frerichs.* It will often be difficult to recognize during life this cause of jaundice, but the following characters may sometimes be of assistance in diagnosis:—

1. A history of some of the usual causes of peri-hepatitis, such as simple ulcer of the stomach, inflammation of the right pleura, general peritonitis, other diseases of the liver, or constitutional syphilis.

2. An antecedent history of the symptoms of peri-hepatitis, and more particularly of acute pain and tenderness in the right hypochondrium with more or less pyrexia.

3. The concurrence of symptoms of chronic atrophy of the liver and signs of portal obstruction (see p. 252).

4. The absence of any history of biliary colic, or of any indications of cancer.

5. The fact of the jaundice, when once it appears, being permanent, not intermittent.

3. *Closure of Orifice of the Duct in consequence of an Ulcer in the Duodenum.*

This is another cause of mechanical jaundice of which the recognition during life is often very difficult. Simple ulcers, like those of the stomach, occasionally form in the duodenum and may, like them, end in hæmorrhage or perforation. Occasionally it happens that one of these ulcers is situated at the part of the

* Op. cit. vol. i. p. 151.

duodenum corresponding to the opening of the bile-duct, and this becomes obstructed by inflammatory products which are apt to become organized, and then the obstruction is permanent. A like result may ensue from the end of the duct being involved in the cicatrix of a duodenal ulcer, as happened in the case of James B. who died in this hospital (Case LXI.). In the diagnosis of this cause of obstruction we must be guided by—

1. The circumstance of the jaundice and other signs of obstruction of the bile-duct being preceded by the symptoms of ulcer of the duodenum, such as pain felt only two or three hours after a meal, when the food is passing from the stomach into the duodenum, with occasional attacks of sudden and profuse hæmorrhage from the stomach or bowels. These symptoms, however, are often absent in cases of ulcer of the duodenum, which may indeed run such a latent course that its existence is not suspected until the occurrence of fatal perforation.*

2. In any case of persistent and intense jaundice with complete disappearance of bile from the motions, the very absence of all symptoms antecedent to those of obstruction of the bile-duct, while at the same time

* In the ninth volume of the 'Pathological Transactions' (p. 197), I have recorded the case of a large finely developed man who died suddenly of peritonitis from a perforating ulcer of the duodenum, and who up to the time of his fatal attack had enjoyed excellent health, and had never suffered from vomiting or even pain after meals. Similar cases have been related by Dr. Budd, in his Lectures on Diseases of the Stomach (p. 149), and by other writers.

there is no evidence of a tumour, of ascites, or of the cancerous cachexia, and no history of biliary colic, will lend some probability to the view that the cause of obstruction is a duodenal ulcer (see Case LXIV.). There is, however, a source of fallacy in the fact that an ulcer of the duodenum, near the opening of the common duct, has been known to induce attacks of spasmodic abdominal pain followed by jaundice. In such a case an accurate diagnosis from gall-stones may be impossible; for, though the occurrence of the paroxysms immediately after obvious errors in diet, or in conjunction with the symptoms of duodenal ulcer, already referred to, might point to this as the probable cause, yet duodenal ulcer, as I have told you, is often a remarkably latent disease. Fortunately cases of this sort are so rare as not often to embarrass the diagnosis.

4. *Stricture from Cicatrization of Ulcers in the Bile-Ducts.*

Stricture or obliteration of the common bile-duct may result from cicatrization of ulcers on its inner surface, produced by the pressure and irritation of gall-stones, or independently, and the impediment to the flow of bile will cause jaundice. When a gall-stone becomes impacted in the common duct, it may lead to adhesions and permanent closure of the duct below it; at other times the gall-stone after causing ulceration escapes, and a stricture forms during the cicatrization of the ulcer; and occasionally ulceration of the bile-duct, with subsequent cicatrization, appears to be in-

dependent of gall-stones.* Most writers on jaundice have referred to stricture of the bile-duct as a possible cause, and you will find two cases in illustration recorded in the 'Pathological Transactions,' one by Dr. Bristowe,† and the other by Mr. Holmes.‡ In the former the stricture was situated in the duct of the left lobe, and in the latter it was in the common, before its junction with the cystic, duct. In both cases the stricture exactly resembled an urethral stricture, and was attended by thickening of the parietes with evident marks of cicatrization; in both cases there was great dilatation of the ducts on the liver side of the constriction; and in both there was jaundice. Gall-stones were found in neither case, and in Mr. Holmes's case there was no history of biliary colic, or of any previous attack of jaundice.

As to the distinguishing characters of this jaundice it may be said —

1. That in many cases there will be an antecedent history of the passage of gall-stones. In all cases where the symptoms of gall-stones are followed by permanent jaundice it may be suspected that the gall-stone has produced an organic stricture or closure of the duct.

* Ulceration of the biliary passages, independent of gall-stones, is occasionally found after death from pythogenic or enteric fever. In my work on the 'Continued Fevers of Great Britain' (pp. 508, 556), I have related a case of enteric fever where fatal peritonitis was excited by a perforating ulcer of the gall-bladder. Frerichs (Op. cit. vol. ii. p. 456) refers to a case reported by Dance, where the ductus communis was found ulcerated, independently of either gall-stones or a specific fever.

† Vol. ix. p. 222.

‡ Vol. xi. p. 130.

2. That when the ulceration of the bile-duct is independent of gall-stones the diagnosis will usually be doubtful. The symptoms of ulceration of the bile-duct have not yet been carefully recorded and analysed, but you will remember that this lesion sometimes gives rise to pyæmia with multiple abscesses in the liver (see pages 151 and 158); and independently of pyæmia it would seem that occasionally, as in Mr. Holmes's case above referred to, the ulceration is ushered in with chilliness and rigors, and is accompanied by pain or uneasiness in the hepatic region.

5. *Spasmodic Stricture of the Bile-Duct?*

When the common bile-duct becomes constricted or obliterated from any of the causes just mentioned, the jaundice is deep and permanent, there is progressive emaciation, and death sooner or later is the result. But it was formerly imagined* that temporary jaundice might result from spasmodic constriction of the duct, constituting what was called *icterus spasmodicus*; and indeed all cases of jaundice where no mechanical obstruction to the flow of bile could be found after death were at one time explained in this way. The contractility of the bile-ducts has been demonstrated by experiment, when they have

* See for example Saunders's 'Treatise on the Structure, Economy, and Diseases of the Liver, and on Bile and Biliary Concretion,' 3rd ed. 1803, p. 100; and Sir Thomas Watson's Lectures on Medicine, 3rd. ed. vol. ii. p. 557.

been mechanically irritated or galvanized in an animal just dead, and it is very possible that during life the passage of irritating bile may cause spasmodic contraction of the duct with severe pain, in the same way as spasm of the bowel is believed to cause colic, and spasm of the bronchi asthma. It is very doubtful, however, if jaundice ever results from spasmodic contraction of this sort. I have already told you (p. 285) that even mechanical obstruction of the duct takes a day or longer to produce jaundice of the integuments, and it is difficult to conceive that a spasmodic stricture, independent of any mechanical obstruction, could last sufficiently long to produce a like result. I have also explained to you how jaundice may be developed independently of any impediment to the flow of bile, so that it is unnecessary to have recourse to the theory of spasm to account for those cases where no mechanical obstruction of the bile-duct can be found after death.

IV. OBSTRUCTION BY TUMOURS CLOSING THE ORIFICE OF THE DUCT OR GROWING IN ITS INTERIOR.

The channel or opening of the bile-duct is liable to become obstructed by cancerous and other growths in the duodenum, by tumours in the pancreas, gall-bladder, or adjacent parts, penetrating the part of the duodenum where the duct opens, or the duct itself in any part of its course; or in rare cases by growths originating in the walls of the bile-ducts themselves.

In Case LXII. there was a cancerous tumour in the head of the pancreas, but the cause of the jaundice was an independent growth in the bile-duct. Another case of jaundice from obstruction of the bile-duct by a tumour developed in its walls is recorded by Dr. Bristowe, in the ninth volume of the 'Pathological Transactions' (p. 220). Case LXIII. is an example of cancer of the pancreas, involving in its growth the gall-bladder and the bile-duct; and you will find two similar cases recorded by Frerichs, in which the cancer had invaded the duodenum and obstructed the orifice of the bile-duct.* The chief characters on which we must rely for the diagnosis of this cause of jaundice are as follows:—

1. Before the jaundice appears, the patient usually complains for some weeks, or longer, of pain, more or less severe, and sometimes lancinating, in the region of the duodenum. This pain continues after the appearance of jaundice, and is usually aggravated two or three hours after taking food.

2. In most cases there are nausea and a tendency to vomit, especially after food.

3. Hæmorrhage from the stomach or bowels is occasionally met with when there is cancerous ulceration of the duodenum.

4. In most cases a hard and tender tumour can be felt more or less distinctly, on careful examination through the abdominal parietes.

* Op. cit. vol. i. Cases VI. and VII.

5. The jaundice, when once it appears, gradually increases in intensity and lasts till death, which, it is important to add, occurs in most cases within four or five months of the first appearance of yellowness of the skin. The very circumstance of jaundice lasting in any case more than six months would be an argument against its being due to a cancerous tumour originating in the wall, or encroaching upon the channel of the bile-duct. This is one reason, in fact, why we have excluded a cancerous tumour from the diagnosis in the case of William M——, now in Cambridge Ward (Case LXIV.).

6. Both before and after the appearance of jaundice there will be progressive emaciation and debility, and the other phenomena of the cancerous cachexia. The diagnosis may also sometimes be aided by indications of cancer in other parts of the body, or by a family history of cancer.

V. OBSTRUCTION BY PRESSURE ON THE DUCT FROM WITHOUT.

There are various tumours and other morbid conditions of the abdomen, which may compress the bile-duct from without, so as to interrupt the flow of bile and cause jaundice. The duration of the jaundice and the prognosis as to the result will depend on the compressing cause in each case.

1. *Tumours projecting from the Liver itself.*

Diseases of the glandular tissue of the liver, even when far advanced, do not as a rule cause jaundice. We have already seen that the secreting structure may be almost, if not quite, destroyed, without any jaundice resulting. Outgrowths, however, from diseased livers, may, like other tumours, compress the bile-duct and interfere with the flow of bile, and thus it is that jaundice may appear in cancer of the liver,* hydatids, and especially the multilocular variety (see page 213), tropical abscess, &c., where as a rule it is absent. The diagnosis of the cause of jaundice in these cases must be based on the presence of the characters of the primary disease of the liver, which have been described to you in previous lectures.

2. *Enlarged Glands in the Fissure of the Liver.*

The lymphatic glands in the fissure of the liver, when enlarged from cancerous, waxy, or tubercular deposit, may compress the bile-duct so as to narrow or obstruct its channel and cause jaundice.† In a large proportion of the cases of cancerous or waxy liver, where jaundice is observed, it is produced in this way. I may, for instance, recall to your recollection the case of Hannah C—— (Case XLI. p. 196), who died

* See a case reported by Dr. Bristowe, in the ninth volume of the Pathological Transactions, p. 223, Case IV.

† See a case recorded by Dr. Handfield Jones in the Pathological Transactions, vol. v. p. 149.

of cancer of the liver and ovary, and whose jaundice was due to compression of the bile-duct by a mass of enlarged glands and dense areolar tissue in the portal fissure. In cases also of primary cancer of the stomach cancerous matter is often infiltrated into the lesser omentum, and may compress the bile-duct and produce jaundice. The recognition during life of the cause of obstruction of the bile-duct in these cases must depend mainly on—

1. The signs and symptoms of waxy disease or cancer of the liver, which I have described to you in former lectures, or of general tuberculosis or cancer of the stomach.

2. The co-existence in most cases of ascites, owing to the pressure being exerted on the portal vein as well as on the bile-duct (see Case XLIII. p. 200).

3. *Tumour of the Stomach.*

A cancerous tumour of the pyloric end of the stomach may cause jaundice by mere compression of the bile-duct. More commonly the duct is compressed by secondary cancerous deposits in the lesser omentum, or in the glands of the portal fissure. The cause of the jaundice in such a case may be recognized—

1. By the fact of its being preceded and accompanied by the ordinary symptoms of cancer of the pylorus, and more particularly by pain and vomiting after food, coffee-ground vomit, and very rapid emaciation.*

* See a case reported by Dr. Bristowe, in the ninth volume of the Pathological Transactions, p. 225, Case VII.

2. By the situation of the tumour, and the fact of its being often accompanied by great dilatation of the stomach, distinguishable through the abdominal parietes.

4. *Tumour of the Pancreas.*

A tumour of the pancreas may not only, as we have seen, invade the duodenum and close the orifice of the bile-duct, or penetrate and obstruct the duct at different parts of its course, but when large, it may compress the duct from without so as to constrict or obliterate its channel. The symptoms of this form of obstruction of the bile-duct will not differ much from those of obstruction from a cancerous tumour of the duodenum (see p. 338), viz.

1. Pain referred to the situation of the pancreas.
2. Nausea and tendency to vomit.
3. A distinct hard tumour at the seat of pain.
4. Jaundice permanent until death.
5. Rapid emaciation and other indications of the cancerous cachexia.
6. The passage of a large quantity of fatty matter in the stools (see *ante*, p. 289).

The phenomena arising from obstruction of the bile-duct by a cancerous tumour in the pancreas may be closely simulated by an abscess in the pancreas, secondary to a simple ulcer of the duodenum, which involves and obstructs the opening of the bile-duct in the manner already described (see p. 332). This appears to me to have been the pathology of a case

recorded by Dr. G. Harley, in the thirteenth volume of the 'Pathological Transactions' (p. 119).

5. *Tumours of the Kidneys.*

Great enlargement of the kidneys, according to Dr. Copland,* may cause jaundice by the pressure exerted by the tumour on the bile-duct; but jaundice from this cause must be extremely rare, as I have been unable to find a case in illustration recorded in the 'Pathological Transactions,' or elsewhere, and I have frequently known the kidneys enormously enlarged from various causes without any jaundice resulting. In a former lecture I called your attention to a case where there was an enormous cystic tumour of the right kidney (Case XXIII. p. 115), containing at least 200 ounces of fluid; and I show you here a cancerous tumour of the left kidney, from a boy aged 8, which weighed 496 ounces and filled almost the whole abdomen.† In neither of these cases was there jaundice.

The diagnosis of jaundice from the pressure of an enlarged kidney on the bile-duct must rest on—

1. The situation and relations of the tumour; and
2. Alterations in the character of the urine.

You will remember, however, that in the case of the enormous cystic tumour of the right kidney just referred to, there was no change of importance in the urine, and in cancer of the kidney the urine may for a long time be free from blood and albumen.

* Dictionary of Medicine, vol. ii. p. 302.

† The details of this case were published by Dr. Vanderbyl, in the Pathological Transactions, vol. vii. p. 268,

6. *A Retro-peritoneal or Omental Tumour.*

A tumour originating behind the peritoneum, and growing forwards into the abdomen, may ultimately involve and compress the bile-duct and cause jaundice; and a tumour originating in the omentum may lead to a like result.* The bile-ducts passing through the morbid growth are compressed and narrowed, and rendered completely impervious. These tumours are usually cancerous, and by the time that they are large enough to compress the bile-duct their existence is sufficiently obvious. The chief difficulty in diagnosis will arise in determining the origin of the tumour. It may be difficult, e.g., to distinguish an omental tumour in the vicinity of the liver and compressing the bile-duct from a tumour of the liver itself; and, indeed, for the solution of the question we must trust entirely to its history and mode of growth. Unfortunately, so far as prognosis and treatment are concerned, the precise origin of the tumour matters little. No known treatment can prevent, or even defer, the fatal event.

7. *An Abdominal Aneurism.*

In very rare cases of aneurism of the abdominal aorta, when the tumour is very large, it may compress the bile-duct and cause jaundice. Dr. Hutton, for instance, has recorded a case where the tumour reached from the crest of the ilium to the lower end of the scapula, and caused jaundice. You will find it referred

* See a case of cancer of the lesser omentum, recorded by Dr. Bristowe, in the *Pathological Transactions*, vol. ix. p. 225.

to in Dr. Stokes's classical work on 'Diseases of the Heart and Aorta.'* But in aneurism of certain of the branches of the abdominal aorta jaundice is much more common, and it is particularly liable to be produced by aneurisms of the hepatic artery. Frerichs has collected four cases† of aneurism of the hepatic artery from different sources, from which it would appear that although the lesion is rare, it has well-defined characters during life. These are mainly—

1. Symptoms of imperfect duodenal digestion, pain in the duodenum and its vicinity occurring two or three hours after taking food.

2. Paroxysms of acute neuralgic pain in the region of the liver, simulating the colic of gall-stones, and due, no doubt, to the pressure of the aneurism on the hepatic plexus of nerves.

3. Persistent jaundice from compression of the bile-duct.

4. Attacks of hæmatemesis and bloody stools, and great anæmia in consequence.

5. A tumour in the right hypochondrium, which may displace the liver upwards. The nature of the case would be rendered still more certain by detecting in this tumour pulsation and a single or double bellows-murmur. In a case, however, recorded by Dr. Stokes, there was no pulsation.

In three of the four cases collected by Frerichs the

* The Diseases of the Heart and Aorta, 1854, p. 633.

† Frerichs, *Op. cit.* vol. ii. p. 378. Frerichs makes five cases, but one of the five, quoted from Stokes, as observed by Dr. Beatty, was an aneurism of the aorta, not of the hepatic artery.

aneurism burst before death—twice into the abdominal cavity, and once into the gall-bladder.

Similar symptoms have been noticed in cases of aneurism of the superior mesenteric artery, although in this form of aneurism jaundice is less common, and hæmorrhages perhaps more so. There was no jaundice in the patient from whose body I removed this specimen some years ago—a man aged 42, who died in this hospital on September 27th, 1860, of profuse hæmorrhage from the stomach and bowels, in consequence of the rupture of an aneurism of the superior mesenteric artery into the duodenum. Neither was jaundice noted in any of three cases of aneurism of the superior mesenteric artery, recorded in the ‘*Pathological Transactions*.’* Two cases, however, of superior mesenteric aneurism are related by Dr. J. A. Wilson, in the ‘*Medico-Chirurgical Transactions*,’† in one of which large quantities of blood were vomited, while the other ‘by pressure on the hepatic apparatus during life had induced jaundice.’ A very interesting case also is recorded by Dr. W. T. Gairdner, where jaundice was produced by an aneurism of the superior mesenteric artery, which opened into the duodenum 22 months before death, and caused repeated and very copious hæmatemesis, with symptoms closely resembling those of gastric ulcer. From this case Dr. Gairdner concluded: ‘that the combination of jaundice with symptoms indicating imperfect duodenal

* Dr. J. W. Ogle’s case, vol. viiii. p. 168; Mr. Holmes’s case, vol. ix. p. 172; and Dr. Wilks’s case, vol. xi. p. 44.

† Vol. xxiv. p. 221.

digestion (cardialgia, pain and vomiting some time after taking food) should in all cases lead to the strong suspicion of a tumour pressing on the ducts of the liver and pancreas, near their duodenal termination; that the co-existence of these symptoms, with fixed pain or oppression at the epigastrium, pulsation in the same region, and hæmatemesis, would very probably indicate aneurismal tumour, even in the absence of more unequivocal signs.*

8. *Accumulation of Fæces in the Bowels.*

Great accumulations of hardened fæces in the bowels may also compress the bile-duct, so as to cause jaundice, and lead to serious errors in diagnosis, hardened scybala being supposed to be nodules of cancer. Dr. Bright has recorded several instances of fæcal accumulation in the colon, mistaken for enlargement of the liver or malignant tumours, and in one of them there was jaundice which disappeared after free evacuation of the bowels.† Frerichs also relates a case where an enlargement of the abdomen from fæcal accumulation was at first ascribed to a pregnant uterus, and subsequently, on the supervention of deep jaundice, to an enlarged liver, but where purgatives dispelled the patient's anxiety about a diseased liver and at the same time her hopes of a child.‡ Errors in diagnosis from this cause are all the more likely to arise, inasmuch as great fæcal accumulation may occur, notwithstanding that the bowels have acted

* Clinical Medicine, 1862, p. 504.

† Abdominal Tumours. Syd. Soc. ed. p. 243.

‡ Op. cit. vol. i. p. 69.

daily or even been relaxed. They may be avoided, however, by attention to the following rules:—

1. On careful manipulation, the doughy consistence and irregular outline of the fæcal mass will often distinguish it from all other abdominal tumours.

2. In all cases of doubt, the judicious use of laxatives and enemata will get rid both of the tumour and of the jaundice.

9. *A Pregnant Uterus.*

Cases have been frequently observed where the presence of a pregnant uterus, often in conjunction with constipated bowels, has caused jaundice, the course of which will be recognized by its appearing in the advanced stages of pregnancy, and disappearing after parturition.

10. *Ovarian and Uterine Tumours.*

Tumours of the uterus and ovary have, in rare instances, been known to compress the bile-duct and cause jaundice. It is sufficient here to mention the fact, as the diagnosis of these diseases from other causes of obstruction of the bile-duct can seldom be difficult.

Treatment of Jaundice from Obstruction of the Bile-Duct.

The treatment for jaundice arising from obstruction of the bile-duct may be considered under two heads, viz.—

A. Those measures which are calculated to remove the obstruction ; and

B. The remedies which are most likely to alleviate the effects of the obstruction.

A. The measures to be adopted for the removal of the obstruction must depend on its nature. Some of the causes of obstruction are removable, while others are not. It may be well therefore to refer to the several causes of obstruction in succession, and first with regard to—

a. Gall-stones.

Under this head we have first to consider what are the best means for expediting the passage of the stone and preventing its impaction, for the longer time the calculus occupies in its passage, the more likely is it to produce ulceration and stricture of the bile-duct, and become permanently arrested ; and secondly, to enquire if there are any remedies which have the power of obviating the formation of fresh concretions, or of dissolving those which already exist in the gall-bladder.

I. Measures for expediting the passage of the Gall-stone.

1. When, from the symptoms which I have already described to you, there is reason to believe that a gall-stone is passing along the bile-duct, it will be well when possible to put the patient in a warm bath, and in all cases to apply heat locally in the form of warm fomentations and poultices.

2. If there be much tenderness on pressure, great relief will often be obtained from the application of a few leeches over the region of the gall-bladder.

3. Along with these measures you must have recourse to full and repeated doses of opium or morphia. The subcutaneous injection of morphia is particularly adapted to cases of this sort, from the rapidity with which it takes effect, and from the irritability of the stomach, which often leads to the rejection of all remedies taken by the mouth. A quarter of a grain of morphia may be injected beneath the skin of the arm, and the operation may be repeated from time to time according to its effect.

4. Belladonna is another antispasmodic, which on several occasions I have found to be of marked utility; half a grain of the extract may be given every two or three hours.

5. Chloroform and ether given by the mouth, or in the form of inhalation, have also been found to be most efficacious; and they possess this advantage, that while they relieve pain, diminish spasm, and are rapid in their action, as in the case of the uterus in parturition, they do not interfere with that muscular contraction which probably assists in the onward propulsion of the stone.

6. Immediate relief is often afforded by large draughts of hot water, containing from one to two drachms of bicarbonate of soda to the pint. According to Dr. Prout, who first recommended this plan of treatment, 'the alkali counteracts the distressing

symptoms produced by the acidity of the stomach, while the hot water acts like a fomentation to the seat of pain. The first portions of water are commonly rejected almost immediately; but others may be repeatedly taken, and after some time it will be usually found that the pain will become less, and the water be retained. Another advantage of this plan of treatment is, that the water abates the severity of the retching, which is usually most severe and dangerous where there is nothing on which the stomach can react. This plan does not supersede the use of opium, which may be given in any way deemed most desirable; and in some instances a few drops of laudanum may be advantageously conjoined with the alkaline solution, after it has been once or twice rejected.*

7. Vomiting of the food which is in the stomach does not require to be checked, but when there is frequent and severe retching, attended with pain, its continuance will lower the vital powers, and increase the danger of rupture of the distended gall-bladder or bile-ducts, and must be checked by effervescing draughts, hydrocyanic acid and ice. For the same reason emetics, which were formerly given with the object of hastening the passage of the concretion, are now justly discarded in the treatment of gall-stones.

8. Strong purgatives also are of little use in expelling the stone, and will exhaust the patient; but if

* On the Nature and Treatment of Stomach and Urinary Diseases, 3rd ed. 1840, p. 263.

the bowels be confined, they ought to be emptied by large enemata of warm water; and when the symptoms make it probable that the stone has escaped into the duodenum, purgatives and copious injections of warm water will hasten the discharge of both it and the accumulated bile.

9. Antimony was long ago recommended by Dr. Bright, with the object of relaxing spasm, but it is apt to increase the sickness, and add to the exhaustion which is the patient's main danger.

II. *Measures for dissolving or preventing the formation of Gall-stones.*

1. There are certain remedies which are believed to have the power of preventing the formation of fresh stones, or even of dissolving those already existing in the gall-bladder. A combination of ether (three parts), and turpentine (two parts), proposed by Durande, a physician of Dijon, in France, for a long time enjoyed a reputation on the Continent for this purpose; and within the last few years, another French physician, Bouchut, has claimed the same virtue for chloroform administered internally. But although both chloroform and ether will dissolve cholesterine, which is the main constituent of gall-stones, out of the body, neither can reach the gall-bladder in a sufficiently concentrated form to accomplish this object during life, and the good effects which were thought to follow their use must be ascribed to their antispasmodic properties.

2. It is very possible, however, that gall-stones can be dissolved. Concretions are occasionally seen whose surfaces exhibit unmistakable signs of erosion. The remedies which are believed to possess this power in the most marked degree are alkalies and diluents. You will do well then to put your patients who have suffered from gall-stones upon an alkaline treatment, and to administer the salts of soda and potash, such as the bicarbonate and phosphate of soda, the latter of which is contained in bile, largely diluted, or the muriate of ammonia with taraxacum; or what is still better, when practicable, send them to drink the alkaline mineral waters of Vichy, Ems, Eger, Carlsbad, or Marienbad. Although it must be confessed that the evidence of the efficacy of mineral waters and alkalies in dissolving or expelling gall-stones is inconclusive and must remain so, there can be little doubt that they improve the general health, and produce such changes in the bile, as lessen the chances of the formation of fresh concretions. In dogs, for instance, with biliary fistulæ, the mere drinking of large quantities of water will increase the amount of water in the bile, and there is evidence that the quantity of soda in the bile may also be increased by taking it into the stomach.

3. In all cases it will be necessary to attend to the patient's digestive and general health. Small doses of blue pill are sometimes very useful. According to Dr. G. Budd, no medicine, in some cases, does such signal good. 'It seems to increase the quantity of

bile, and at the same time to render it more healthy, and certainly often improves in a striking manner the general health.*

4. Lastly, it will be necessary to counteract those habits on the part of the patient, which experience has shown to conduce to the formation of gall-stones. The patient must rise early and take plenty of exercise in the open air, sleep in an airy bed-room, live sparingly, and avoid fat and malt liquors.

b. Hydatids, Distomata, and other foreign Bodies in the Bile-Ducts.

These causes of obstruction of the bile-duct must be treated on the same principles as gall-stones, with anodynes and antispasmodics. The bursting of an hydatid tumour into the bile-duct is usually preceded by more or less peritonitis, and followed by inflammation of the hydatid, which will call for absolute rest, leeches, warm fomentations and opiates. (See Cases IX. and X. pp. 90, 94.)

c. Inflammation of the Bile-Ducts.

The treatment for obstruction of the bile-duct by inflammatory obstruction of the lining membrane with exudation in the interior has been considered in a former lecture (p. 134).

* Op. cit. p. 387.

d. Organic Stricture and Tumours of the Bile-Duct.

For the various forms of organic obstruction of the bile-duct arising from stricture or obliteration of the duct or from tumours growing in its interior, no treatment is likely to be of any avail. The obstruction is irremovable, and the jaundice is permanent. In cases, however, where there has been a history of syphilitic peri-hepatitis, mercury and iodide of potassium deserve a trial.

e. Pressure on the Bile-Duct from without.

When the obstruction is due to pressure on the duct from without, the treatment must vary according to the compressing cause. Some of these causes are removable; others are not. When the pressure is due to an abscess or hydatid of the liver, or to an ovarian cyst, it will be removed on evacuation of the fluid contents of the tumour; but the pressure of cancerous nodules projecting from the liver, enlarged cancerous glands in the portal fissure, tumours of the stomach, pancreas, kidney, omentum, and uterus, and of abdominal aneurisms, cannot be influenced by treatment. When the symptoms point to waxy or tubercular glands in the fissure of the liver as the cause of pressure, improvement may sometimes be observed to follow the use of iodide of potassium, iron, nitro-muriatic acid, and cod-liver oil. Fæcal accumulations in the colon are to be got rid of by castor oil, the administration of frequent small doses of

extract of belladonna, and copious oleaginous or warm water enemata. Lastly, when jaundice shows itself during pregnancy, care must be taken to ascertain whether the pressure of the gravid uterus be not aggravated by accumulation of fæces in the bowels.

B. In the next place we have to consider what are the most suitable remedies for relieving the effects of irremovable or permanent obstruction of the bile-duct.

1. One of the first effects of complete obstruction of the bile-duct, if it be not speedily removed, is the accumulation of bile in the bile-ducts and gall-bladder, which become greatly distended and sometimes inflamed in consequence (see ante, p. 142), and under these circumstances advantage will often be derived from leeches to the right hypochondrium or round the anus, warm poultices, laxatives, diuretics and diaphoretics, and from taking as little fluid as possible in the way of drink.

2. The diet in all cases requires careful regulation. It ought to be easy of digestion and mainly nitrogenous. Oleaginous and saccharine matters and malt liquors ought to be for the most part excluded.

3. The bowels will require attention. In most cases they are constipated, and laxatives will be necessary, and of these the best is a combination of the compound colocynth or rhubarb pill (gr. vi) with blue pill (gr. ij), and extract of henbane (gr. ij). No good can be expected from the use of mercury in such cases; but there can be no objection to the use of

both mercury and podophyllin, as an occasional purgative. Practically their use in moderation is not attended with those injurious consequences which have been theoretically ascribed to them. Although under ordinary circumstances they produce bilious stools, there is no evidence, as I have already pointed out to you, that mercury increases the amount of bile secreted by the liver (p. 309).

4. Flatulence and other dyspeptic symptoms will in many cases call for treatment. The flatulence will often be relieved by the ethers and essential oils, the gum-resins of assafoetida and galbanum, and by vegetable charcoal; but in most cases the best remedies are those which have antiseptic properties. Bile is an antiseptic, and its withdrawal from the bowels entails decomposition of their contents with generation of gas, but this decomposition will be prevented by the use of such remedies as creasote, turpentine and carbolic acid (see p. 259). Flatulence and other dyspeptic symptoms arising from the want of bile in the bowels are also often greatly relieved by the use of purified bile from the ox or pig, which may be given in doses of from three to six grains about two hours after meals. As it is not desirable that the bile should come in contact with the stomach it is well to give it enclosed in capsules, or in pills coated with a solution of tolu in ether. The choleate of soda, of which ten grains may be taken in peppermint water, has also been found useful for the same purpose. The alkalies and mineral acids (p. 127), but more commonly the

former, in conjunction with calumba, or with taraxacum, chiretta, gentian, or quinine, are also often of use for improving the appetite and digestion.

5. In all cases of jaundice from obstruction of the ducts it is important to attend to the functions of the kidneys and skin. The kidneys are the principal channels by which the accumulated bile is got rid of from the system, and any disease of these organs (as in Case XXIX. p. 137, and Case LI. p. 263) will add greatly to the patient's danger. Persons suffering from jaundice due to obstruction of the bile-duct must avoid sudden chills, and will be benefited by occasional warm baths and by the use of diaphoretics and diuretics.

6. The itchiness which is often a source of much distress will sometimes be alleviated by warm baths, the use of a flesh-brush, and the internal administration of bicarbonate of potash (see p. 291). Recourse also must be had to opiates and other anodynes to procure sleep; but all treatment will sometimes fail to give much relief, as we found in the case of William M—— (Case LXIV.).

7. When there is great debility, or when the patient suffers from boils or carbuncles, improvement will sometimes follow the use of quinine or iron, and it will be necessary to allow small quantities of alcoholic stimulants. Of these the best are hock, dry sherry, sound claret, and diluted brandy or gin.

8. When cerebral symptoms supervene, the treatment which has been found most efficacious consists

in blisters to the nape or scalp and purgatives. It will be well also to act on the skin by means of diaphoretics, the warm bath, or, what is better, by the hot air bath, and if there be no albumen in the urine, to give diuretics.

9. Occasionally, the treatment may have to be modified in accordance with symptoms arising from the disease to which the obstruction of the bile-duct is due, as, for instance, in cases of cancer of the stomach, duodenum or pancreas, or of abdominal aneurism.

10. Lastly, it is well to remember that in those cases where you succeed in removing the obstruction, the jaundice of the skin and conjunctivæ may persist for a considerable time afterwards, and that then its departure will be expedited by warm baths, diaphoretics, purgatives and diuretics, and also by benzoic acid, which may be given in doses of four grains made up into two pills with a little glycerine, three times a day.

I shall now proceed to recall to your recollection the particulars of a few cases of jaundice from obstruction of the bile-duct, which, for the most part, have been under your observation in the wards.

The first case is a good illustration of enlargement of the liver and jaundice from gall-stones. Many of you had an opportunity of seeing the patient, who attended regularly as an out-patient under my care from Sept. 1865, to July, 1866.

CASE LVIII.—*Jaundice from Obstruction of the Bile-Duct by Gall-stones.*

Jacob G—, aged 59, came to the hospital on September 13, 1865, complaining of dyspepsia, flatulence, and jaundice. His illness had commenced a week before with a paroxysm of rigors and excruciating twisting pain in the abdomen, accompanied by vomiting. The paroxysm lasted two or three hours, and had recurred several times during the week. The jaundice was intense; the urine was of the colour of porter and contained much bile-pigment; and the patient complained greatly of a bitter taste in the mouth and of a ‘nasty stinging itchiness’ of the entire skin. The motions were clay-coloured, and the hepatic dulness was uniformly increased, amounting to upwards of five inches in the right mammary line. No tumour corresponding to the gall-bladder could be felt, and there was but slight tenderness below the right ribs.

By October 6, the jaundice had quite disappeared; but on the 27th he had a return of the severe paroxysms of pain with jaundice, and all the other symptoms as before. This ran much the same course as the first attack; and between October, 1865, and July, 1866, he had five other attacks, always accompanied by vomiting and by intense jaundice, which passed off in the intervals. He was not aware of having voided any gall-stones after the attacks, but being an out-patient there was no opportunity of examining the motions. When I last saw him in July, 1866, he had no jaundice; he had been free from any paroxysm of pain for six weeks; he was not emaciated, and his general health was tolerably good.

The treatment which seemed to be of most service in this case consisted in alkalies (bicarbonate of soda), taraxacum, and vegetable bitters, and the use of opium, warm fomentations and warm baths during the paroxysms of pain.

In the next case also there could be little doubt from the symptoms that the jaundice resulted from obstruction of the common duct by a gall-stone or inspissated bile. If a gall-stone, the patient may possibly

have voided it before admission, as the appearance of the urine when she was first seen, made it probable that the jaundice was already receding. The circumstance of the itchiness of the skin preceding the jaundice and ceasing on its appearance is also worthy of notice (see p. 290).

CASE LIX.—*Jaundice from temporary Obstruction of the Bile-Duct by Gall-stones.*

Mary C——, aged 64, whose mother had died of 'jaundice' at the age of 52, was admitted under my care on March 10, 1868. She had enjoyed good health all her life until five years before admission, when she was laid up for three months with a fracture of the arm and other injuries, and about three years afterwards she was an out-patient at this hospital, suffering from cough and pain in her right side. In June, 1867, she had for the first time an attack of severe spasm in her right side, with vomiting, followed by jaundice of the face and eyes. In November, 1867, and again in January, 1868, she had a similar attack, the jaundice on each occasion disappearing after a week or ten days. The attack for which she was admitted had lasted eighteen days, and commenced with severe pain in the region of the liver, coming on in paroxysms, which would last for five or six hours and were accompanied by violent retching. On February 29, she first noticed that her skin was yellow, and since then the jaundice had increased, but the paroxysms of pain and retching had been much less frequent and severe. At the commencement of her attack, she had suffered much from itchiness of the skin, which had ceased on the appearance of the jaundice.

On the patient's admission, the skin and conjunctivæ were of a deep orange-yellow colour. She complained of nausea, want of appetite, and of a bitter taste in the mouth; the bowels had been confined, but had been kept open by medicine; the last motion two days before admission had been clay-coloured. The urine was acid, and had a distinct reaction of bile-pigment, but was not nearly so dark as might have been expected from the colour of the skin. The right lobe of the liver extended as low as the umbilicus,

but this seemed to be the result of a malformation from tight-lacing; there was considerable tenderness over it on pressure. The pulse was 84 and slightly irregular, and there was a faint systolic bellows-murmur over the left apex of the heart.

The treatment consisted in a warm bath followed by warm fomentations over the liver, saline purgatives, an effervescing mixture containing citrate of potash, and a third of a grain of extract of belladonna three times a day for a week.

The patient had no return of the pain or vomiting after admission, and the motions from the first contained abundance of bile. All the motions passed during the first week after admission were strained through muslin, but no gall-stones were found. On March 23, a warm bath was ordered three times a week. On the 30th, the jaundice had almost disappeared, and on April 6, the patient left the hospital apparently well.

In former lectures I have brought under your notice three other cases of jaundice from gall-stones. In Case XXIX. p. 137, the jaundice resulted from inflammation of the biliary passages excited by gall-stones in the gall-bladder, but there had been no history of biliary colic; in case XXXI. (p. 145) there was jaundice, with enlargement of the liver and gall-bladder, from obstruction of the common duct by a calculus; and in Case XXXV. (p. 158) attacks of biliary colic and jaundice were followed by pyæmic abscesses in the liver, and death.

You will remember also, that when on the subject of enlargements of the liver from hydatid tumour, I related to you two cases where the bile-duct became obstructed by hydatid vesicles which had escaped from the ruptured parent hydatid (Case IX. p. 90, and Case X. p. 94), and that in one of these cases the passage of

hydatid vesicles along the bile-duct produced all the phenomena resulting from the passage of gall-stones.

I have also called your attention to several cases where jaundice resulted from inflammation of the bile-ducts interfering with the flow of bile (Cases XXVII. XXVIII., XXIX., and XXX.).

The next case is an example of a very rare form of jaundice, where the cause was a congenital closure of the bile-duct.

CASE LX.—*Jaundice from Congenital Closure of the Bile-Duct.*

Esther W—, aged 2 months, was brought to me by her mother as an out-patient at this hospital on January 7, 1862. The mother stated that the child had appeared healthy when born, but that a few days afterwards it was noticed to be unusually yellow, and that this yellowness had been increasing, while the child had been suffering from diarrhœa and emaciating. When first seen by me the child was very thin, and the skin and conjunctivæ were of an orange-yellow colour. The motions were perfectly white, and very offensive. A teaspoonful of chalk mixture to be taken two or three times a day was all that was prescribed.

On January 21, the child was not quite so yellow, but it was thinner, and the motions were red like brick-dust, evidently from the presence of blood. It had also had several attacks of slight epistaxis.

During the next week the child continued much in the same state, still losing a little blood occasionally from the nose, and getting gradually thinner, although there was less diarrhœa.

On March 11, it was noticed that several black lumps had appeared over the chest and back. They varied in size up to three quarters of an inch in diameter, and were considerably raised above the surface; they were evidently due to extravasations of blood beneath the skin.

On March 25, the mother stated that the child had been much

worse for the two previous days, vomiting everything she swallowed, and the vomited matters containing blood. The ecchymoses on the surface had also increased, both in number and size.

Two days after this the child died, and on careful dissection the common bile-duct was found to be completely obliterated, its place being occupied by a small quantity of areolar tissue. The gall-bladder was extremely small and collapsed, and contained only a few drops of colourless fluid. The opening of the duct in the duodenum was found with difficulty, but a probe could not be passed through it into the bile-duct. The liver was jaundiced and presented a few fibrous bands of adhesion on its under surface, but in other respects appeared normal. There was altered blood in the contents of the bowels, and several small extravasations beneath the mucous membrane of both the stomach and intestines.

In Case LXXXVIII. (Lect. XI.), the jaundice was the result of constriction of the duct from perihepatitis, and Case LXI., of which I show you here the specimen, dissected by me several years ago while pathologist to the hospital, is a rare example of obstruction of the orifice of the bile-duct, in consequence of its being involved in the cicatrix of a duodenal ulcer. In the case from which this preparation was obtained, there was painful enlargement of the gall-bladder, and jaundice, arising from obstruction of the duct caused apparently by the cicatrization of a duodenal ulcer. The contraction of the liver in this case was no doubt due to the long duration of the obstruction, the hepatic tissue having become atrophied from the pressure upon it of the permanently distended bile-ducts.

CASE LXI.—*Jaundice from Obstruction of the Common Bile-Duct by the Cicatrix of a Duodenal Ulcer—Dilatation of the Bile-Ducts and Atrophy of the Liver.*

James B—, aged 69, a coachman, of small frame and spare habit, was admitted, under Dr. Stewart, on May 4, 1861. During the greater part of his life he had been in the habit of drinking a good deal, but for the last seven years he had been a very steady, sober man. With the exception of an attack of acute bronchitis nine years before, which left him 'asthmatic,' he had enjoyed good health until four months before admission, when he had been suddenly seized with acute pain in the right hypochondrium, vomiting of bitter matter, and much fever. After a fortnight his skin became jaundiced, and he had great formication. The jaundice increased in intensity but the formication diminished. Latterly he had suffered from palpitation in the cardiac region and throbbing in the head, and he had lost both flesh and strength. At the commencement of his illness he had been for a fortnight in another hospital where he had been salivated.

On admission, the patient was emaciated and feeble; the pulse was 72, and intermitting; the whole skin and the conjunctivæ were of a bright lemon-yellow. The patient complained of a dull pain in the region of the liver, the dulness on percussion of which appeared considerably increased, measuring upwards of five inches in the right mammary line. On more careful examination, it was ascertained that this enlargement was limited to the situation of the gall-bladder, and that posteriorly and laterally the hepatic dulness was diminished. The tongue had a yellowish coat. He had no bitter taste, but his sense of taste was in a great measure abrogated; he had no appetite and his bowels were confined, the motions being nearly white. The urine was dark, like porter, and contained much bile-pigment. The bladder had to be evacuated by the catheter. Over both lungs sonorous rhonchi could be heard and the expiration was prolonged. He still complained of itching of the skin at night.

The patient was treated with alkalies, ammonia, vegetable bitters, and stimulants; but his prostration rapidly increased, a bad sore appeared over the sacrum; the tongue became dry and brown and sordes collected on the teeth; the motions became very

dark from the presence of blood ; the pulse rose to 90 ; the signs of bronchitis increased, and for several days before death, which occurred on May 19, there was low muttering delirium.

At the autopsy, the liver was found to be small, pale, and flabby. Its lower margin did not reach so far as the edge of the ribs. The gall-bladder was about four times the normal size, and was filled with a colourless, flaky fluid, destitute of any tint of bile. The cystic, hepatic, and common ducts were all enormously dilated, the common duct being larger than one's finger, and all were filled with a colourless fluid, similar to that in the gall-bladder. The bile-ducts also in the interior of the liver were greatly dilated as far even as the outer surface, and filled with a similar fluid, which flowed out when the liver was cut into. There was no calculus in the gall-bladder or in any of the ducts, but the orifice of the common duct in the duodenum was completely blocked up. The coats of the bowels at this part were dense and considerably thickened, forming a nipple-like prominence about the size of a hazel-nut, and around this the mucous membrane had a radiated, puckered appearance, as if from the cicatrization of an ulcer. There were no ulcers or cicatrices elsewhere in the bowels, and no evidence of morbid deposit in the head of the pancreas or in the adjacent glands, but the pancreatic ducts were much dilated. The secreting tissue of the liver was of an olive-yellow colour, and flabby, but not friable. The outlines of the lobules were obliterated, and on microscopic examination, much granular and oily matter was discovered, but the secreting cells had mostly disappeared. There was much atheroma of the aorta and of the valves of the heart. The lungs presented the characters of bronchitis and emphysema of old standing. The prostate was enlarged, and the bladder contracted, its mucous membrane being very hyperæmic, encrusted with flakes of diphtheritic exudation as large as a shilling ; the kidneys were granular, and the pelves and calices were dilated.

When lecturing on the subject of enlargements of the liver from cancer, I brought under your notice two examples of this disease in which there was jaundice. In one (Case XLIII. p. 200) there was unfortunately no post-mortem examination ; in the other (Case XLI.

p. 196) the jaundice was due to compression of the common duct by a dense mass of areolar tissue and enlarged cancerous glands in the portal fissure.

In the two cases which follow there was a cancerous tumour in the head of the pancreas, but in the one (Case LXII.) you will see by this preparation (Mus. Cat. Series IX. No. 18) that the obstruction of the bile-duct was due to an independent cancerous growth springing from its lining membrane, while in the other (Case LXIII.) it was produced by a mass of cancerous matter in the portal fissure.

CASE LXII.—*Jaundice from Obstruction of the Common Bile-Duct by a Cancerous Growth from its Lining Membrane—Dilatation of the Bile-Ducts and enlargement of the Gall-bladder—Cancerous growth in the Pancreas.**

The patient, from whose body this preparation was obtained, was a cabinet-maker, aged 36, who was admitted into this hospital on September 1, 1857. His illness had commenced on June 1, with vomiting and purging, followed after three or four weeks by intense jaundice, dark urine, and white stools. He had no symptoms of gall-stones, but there was a settled dull pain in the region of the liver, which was enlarged and tender. He complained of loss of appetite, thirst, and lassitude. He had been treated at first with mercurials, and subsequently with nitro-muriatic acid and taraxacum and saline aperients; and by the middle of August, the liver seemed reduced to nearly its normal size and was free from tenderness, but the gall-bladder was enlarged and seemed to be about the size of a small pear, and the jaundice was increased.

* The particulars of this case were recorded by Dr. Vanderbyl, in the Pathological Transactions for 1858, vol. ix. p. 228.

When admitted into the hospital, the patient had intense jaundice, and the urine was loaded with bile pigment of which the *feces* contained not a trace. The liver was of the natural size, but the gall-bladder could be felt reaching as low as the umbilicus, and was the seat of considerable tenderness. There was much tympanitis, but no ascites or enlargement of the abdominal veins. The patient was very emaciated and low-spirited, had no appetite, and passed sleepless nights.

Iodide of potassium, bicarbonate of potash, the mineral acids, taraxacum, gentian, quinine, and nitro-hydrochloric acid baths and lotions were tried in turn, but nothing made any impression on the disease. The tumour corresponding to the gall-bladder increased in size and became more tender, and about three weeks after admission the patient became suddenly unconscious and had symptoms of rapid dissolution with muttering delirium. These symptoms, however, gradually subsided in the course of an hour and did not recur, but the patient gradually sank, and died on October 21.

Autopsy.—The gall-bladder projected 3 inches beyond the anterior margin of the liver, and extended to within an inch of the anterior superior spinous process of the ilium; its length was altogether $7\frac{1}{2}$ inches. Its coats were attenuated, and its inner surface had lost its reticular appearance and was rough, red, and granular. It contained 15 fluid ounces of a pale turbid fluid, like barley-water, which, on standing, deposited numerous small particles of inspissated bile. The fluid was alkaline; its specific gravity was 1010; and it contained numerous very large cells presenting from two to four nuclei with nucleoli, besides flakes of tessellated epithelium, granular matter, &c. The cystic and the greater part of the common bile-duct were also much dilated, the latter being large enough to admit a man's thumb, and on cutting into the liver a large quantity of white fluid like that in the gall-bladder escaped from the biliary ducts, which were very greatly dilated. The liver weighed 60 ounces; it was of a dark olive-green colour, and the outline of the lobules was distinct. On tracing the common bile-duct to the bowel, it may be seen in the specimen to be obstructed by an exuberant growth of medullary cancer growing from its inner surface, and extending for about 2 inches upwards from the duodenum. Before the duct was slit up, a probe could be passed with difficulty from the

duodenal orifice through the obstruction. On cutting into the growth, a glairy juice exuded which contained large compound nucleated cells, similar to those found in the fluid in the gall-bladder, and free nuclei. The head of the pancreas was involved in a growth larger than an orange, which on section presented a medullary appearance, and yielded a copious, thick, milky juice, containing many large nuclei, but no mother-cells. This tumour had compressed the bile-duct, but did not encroach upon its walls, and the growth in the duct appeared to have arisen independently. The heart weighed only 6 ounces. All the organs and tissues of the body, including the brain, were deeply jaundiced, but, with the exceptions mentioned, were in other respects healthy. The contents of the bowels were devoid of bile-pigment.

CASE LXIII.—*Cancer of the Pancreas and of the Gall-bladder—Jaundice from Obstruction of the Bile-Duct.*

At the end of May, 1866, I was consulted in reference to the case of Mr. D—, a gentleman between 60 and 70 years of age, who had jaundice. His illness had commenced about the beginning of January, with symptoms of bronchitis, loss of appetite, and restless nights. On one of the last days in February he had been exposed to severe cold, and the next morning he awoke with intense nausea and retching, which continued till night. Slight febrile symptoms followed; the pulse rose ten beats above its average; the appetite was capricious, and the bowels required aperients. He rapidly lost flesh, and about the middle of April jaundice appeared. His symptoms at the time I was consulted, were as follows:—Deep jaundice of the skin and conjunctivæ. Urine scanty, and very dark, and contained a large quantity of bile-pigment. Slight tendency to diarrhœa, and motions devoid of bile, of a leaden hue and very offensive, but sometimes contained a small quantity of blood from piles. The liver appeared to be quite within its normal limits, but there was occasional uneasiness in the right hypochondrium, and decided tenderness with slight hardness about the situation of the gall-bladder. The superficial veins of the abdomen were also slightly enlarged. The pulse was 72. There were increasing emaciation and debility, with decreasing appetite and occasional retching, and the countenance exhibited an anxious, cachectic aspect.

The treatment consisted in effervescing draughts with hydrocyanic acid and Battley's liquor opii, and in rubbing in externally iodide of potassium ointment.

About the beginning of June, the vomiting became more urgent; almost everything that was swallowed was rejected; the pain increased; the tongue became thickly furred and brown; and the emaciation increased daily. After three days of unconsciousness and low delirium, Mr. D. died on June 19.

A post-mortem examination was made by Mr. Moreton, of Tarvin, Cheshire, to whom I am mainly indebted for the following particulars, and for forwarding to me some of the parts for examination. There were firm bands of adhesion connecting the whole surface of the liver to the surrounding parts. There was no ascites. The gall-bladder was about the size of a hen's egg, its walls were greatly thickened from cancerous deposit, and its cavity contained about a teaspoonful of thick fluid like bloody cream. The opening of the gall-bladder into the cystic duct was closed, and both the cystic and common bile-duct were embedded in, and obstructed by, a mass of cheesy, cancerous matter. There were no cancerous deposits in the liver, but in the head of the pancreas there was a mass of cancer as large as a small orange. All of these deposits gave on section a creamy juice, containing the usual cellular elements of cancer. The mucous membrane presented marked signs of catarrhal inflammation, particularly about the opening of the duct.

The patient whose case I will next relate to you has been under your observation for nearly six months, and you will remember how frequently I have called your attention to the distressing itchiness from which he has suffered. He has also been the subject of boils and carbuncles and of yellow vision (p. 295); but the main source of interest in his case is the cause of the obstruction about which we have often speculated. The improvement which has taken place latterly in his appearance and weight is opposed to cancer of the pancreas or duodenum, or indeed to cancer of any other

organ; and although there has been no clear history of biliary colic, the probability is, as I stated to you on the man's first admission, that the obstruction is due to an impacted gall-stone, to a simple stricture of the duct, or to its orifice having become involved in the cicatrix of a duodenal ulcer. But whatever be its cause, it is to be feared that the obstruction is now permanent. Notwithstanding the improvement that has taken place, the fact that the jaundice is less, coupled with the diminished area of hepatic dulness and the non-appearance of bile in the stools, points to a destruction of the secreting tissue of the liver and is the reverse of favourable.

CASE LXIV.—*Permanent Jaundice from Closure of the Bile-Duct.*

William M——, aged 50, a porter, but formerly a fireman, was admitted under my care on November 26, 1867. Twelve years before admission, he had suffered from bronchitis, and four years before he had been laid up for seven weeks with rheumatic fever. For ten years he had been subject to piles, and had occasionally lost a good deal of blood from them. He had been in the habit of drinking a good deal of beer, but he had never been a spirit-drinker. There was no history of cancer in his family; his father was alive, aged 83, his mother died at 63 from rupture of a blood-vessel. Six weeks before admission, he noticed his urine to be very dark, and his motions pale, and that he was becoming thinner and weaker, and about the same time he began to suffer from itchiness of the skin. After about a fortnight he found his skin becoming yellow, and he was often awoke in the night with severe pain at the pit of the stomach, alleviated by friction, which seemed to bring up a quantity of gas. The skin and urine gradually became darker, and on one occasion, a fortnight before admission, he vomited about a pint of clear watery tasteless fluid.

On admission, the patient was thin, and had very deep jaundice of the entire surface of the skin and conjunctivæ, but his chief complaint was of intense itchiness over his whole body, though most severe in the palms of the hands and soles of the feet, which kept him awake at night. The skin was marked by numerous scratches, but there was no eruption. The urine was very dark like porter, and yielded the reaction of bile-pigment in a marked degree, but contained no albumen. There was no feeling of pain or tenderness in the region of the liver, which did not project beyond the edge of the ribs, and which did not seem to be materially altered in size, the dulness in the right mammary line being $3\frac{1}{4}$ inches and the man being of small build and short stature. Corresponding to the gall-bladder, however, the hepatic dulness seemed to project about half-an-inch from the general boundary-line. The splenic dulness was not increased. There was no ascites, no appreciable abdominal tumour, no enlargement of the abdominal veins, and but slight tympanitic distention of the bowels. The tongue was moist and coated white. He complained of a bitter taste, especially in the morning, and of nausea and loss of appetite; the bowels were regular, and the motions clay-coloured. The cardiac and respiratory symptoms were normal, except that the pulse was only 52. The temperature was 97.5° . His weight on October 2, before he began to be ill, had been 132 lbs.; a few days after admission it was 116 lbs.

He was ordered a mixture with bicarbonate of soda (gr. x), and spirit of chloroform (℥ xx) three times a day, and a henbane draught at night.

A few days after admission, it was ascertained that the patient had yellow vision; everything white appeared to him yellow. The conjunctivæ were at the same time considerably injected. The yellow vision disappeared about the end of December, and did not recur, although no change took place either in the jaundice or in the amount of blood in the conjunctival vessels.

Henbane, Indian hemp, opium, subcutaneous injections of morphia, ox-bile, benzoic acid, and warm baths were resorted to in succession, but failed to relieve the itchiness. He suffered also much from attacks of flatulent colic, which appeared to be relieved by a pill with creosote (℥ j) and galbanum (pil. galb. co. gr. iij), taken twice a day, and subsequently by the confectio terebinthinæ; pills of ox-bile were tried without any result. On

December 13, and again on December 28, he vomited his breakfast.

On December 30, the following note was entered in the case-book:—‘For the first time there is felt what appears to be a deep-seated, hard tumour, about the size of a walnut, $1\frac{1}{2}$ inch above and to the right of the umbilicus, which is not at all tender;’ and on February 10 there is this additional note: ‘Tumour formerly noted is more distinct; it appears to be of about the size of a small orange, and situated to the right of the umbilicus. It is hard, and its surface is slightly lobulated but not at all tender on pressure. It is distinctly movable, and appears to be continuous above with the liver, with which it is connected in precisely the situation of the gall-bladder. Its lower extremity is fully $2\frac{1}{4}$ inches below the general outline of the liver.’

The itching continued to be very intense, and was the source of great distress; but on three different occasions, viz. on January 20, February 8, and March 18, it was at once, and for many days relieved by a mixture containing 10 grains of the bicarbonate of potash with 10 grains of the nitrate of potash taken three times a day. During the last fortnight of February, the patient had many attacks of severe abdominal pain, often lasting for several hours, and relieved by eructations of gas and fluid. On February 26, he vomited his food. His abdomen about this time became moderately distended from tympanitis. From the first his bowels had been open, two, three, or four times a day, the motions being of fair consistence, but clay-coloured and very offensive. The jaundice and the colour of the urine varied in intensity from time to time, the latter being sometimes almost black from the amount of bile-pigment. His appetite failed entirely, but was considerably improved by quinine pills—ordered on February 12. He continued to lose weight until March 4, when he weighed only 105 lbs., being a loss of 11 lbs. since admission; after this his appearance improved somewhat, and on April 1 he had gained 3 lbs. About the end of January he suffered for about a week, from small, but very painful, boils in the meatus of the left ear. At the beginning of March, a crop of small painful boils appeared on the back part of the scalp, and at the end of March, a large carbuncle formed over the occiput and upper part of the neck, on account of which he was transferred for three weeks to one of the surgical wards.

[May 1st, 1868.—His condition now is as follows. His appear-

ance is on the whole improved, and he has gained $5\frac{1}{2}$ lbs. in weight since March 4. The jaundice is decidedly less, and the urine contains less bile-pigment, but the motions are still clay-coloured, without a trace of bile. The itchiness has been more distressing than ever, since taking a mixture containing nitric acid and bark. The tumour has undergone little change since February 10; it is still hard and painless. The area of hepatic dulness is decidedly less than on admission. There is less tympanitis and no ascites. The patient's appetite is better. The bowels are still open two or three times a day.

May 27th.—Has gained 7 lbs. in weight since March 4. Still suffers much from the itchiness, but jaundice is less and urine paler than they have been since admission. For a week has taken three times a day, two hours after meals, a gelatine capsule containing three grains of ox-gall, but the motions are still clay-coloured, and scarcely darker than they were before.

June 6th.—Two days ago had a very severe attack of abdominal pain, lasting for two hours, and accompanied by vomiting, perspiration, coldness of surface, and weak pulse. His general condition remains the same, and there is no change in the colour of the motions.]

LECTURE X.

JAUNDICE.

INDEPENDENT OF OBSTRUCTION OF THE BILE-DUCT.

DIAGNOSIS OF THE CAUSES OF JAUNDICE.

GENTLEMEN,—In my last lecture I explained to you that jaundice in those cases where it is independent of any mechanical impediment to the escape of bile from the liver might be referred to one of the following causes, viz.—

I. Poisons in the blood interfering with the normal metamorphosis of bile.

II. Impaired or deranged innervation interfering with the normal metamorphosis of bile, or increasing its secretion.

III. Deficient oxygenation of the blood, interfering with the normal metamorphosis of bile.

IV. Excessive secretion of bile, so that more is absorbed than can undergo the normal metamorphosis.

V. Undue retention of bile in the biliary passages and bowels, from habitual or protracted constipation.

I purpose devoting the greater part of this lecture to the consideration of jaundice from these various causes.

I. JAUNDICE FROM POISONS IN THE BLOOD.

Cases are not uncommon in which jaundice results from a poisoned condition of the blood, such as that which exists in persons affected with the various specific fevers. It is very probable that when jaundice occurs in such cases, its mechanism is not always precisely the same. Sometimes, as for instance in many cases of ague and relapsing fever, it is associated with considerable enlargement and congestion of the liver, and this congestion is often the chief, if not the sole, cause of the jaundice; and at other times the duodenal orifice of the duct may be plugged by catarrhal inflammation. But in many, and these are much the more serious cases, the jaundice is independent of either congestion of the liver, or of obstruction of the bile-duct: during life there is no lack of bile in the evacuations, and after death the liver is often anæmic rather than congested. In these cases too, the cerebral and other phenomena of the typhoid state are usually prominently developed, and there is reason to believe that here, as in other diseases, this typhoid condition is due not to the presence of bile in the blood (see p. 297), but to imperfect elaboration or elimination of the normal products of metamorphosis in the blood and tissues, of which the jaundice is only a visible sign. Although careful observations on this form of jaundice are still wanting, it has been repeatedly noticed that there is

a diminution of urea in the urine, and that in some instances the urine has been found to contain leucine and tyrosine, which, as I have already told you, are indications of imperfect metamorphosis (see pp. 230, 299). The general condition of the patient, in fact, is very similar to what has been described to you in a previous lecture as occurring in acute or yellow atrophy of the liver (see p. 228), and it is very probable that the pathology of the two conditions is similar. In both there is a morbid condition of the blood, as the result of which the metamorphoses which usually go on in that fluid are impeded or arrested, there is a deficient excretion of urea, and a tendency to the development of leucine and tyrosine in the liver, spleen, kidneys, blood, and urine. In both, the jaundice is probably merely one of the results of this impaired metamorphosis, the bile-pigment absorbed into the blood not being transformed as in health. Occasionally the liver presents an appearance like that of an early stage of acute atrophy, and indeed I have already had occasion to tell you that the poisons of typhus fever, and of other allied diseases must be reckoned among the causes of yellow atrophy of the liver (p. 233).

We may now consider very briefly jaundice as it results from the several blood-poisons, some of which give rise to it much more readily than others.

1. *The Poisons of the various Specific Fevers.*

a. *Yellow Fever.*

The yellow fever of the tropics derives its name from the frequency with which it is complicated with jaundice. It has been demonstrated over and over again that the yellow suffusion of the skin and eye in this disease is occasioned by the presence of bile, which is also found in the urine. Post-mortem examinations, and the fact that in the earlier stages of the disease there is a full supply of bile in the alvine evacuations, have satisfactorily proved that the jaundice is independent of any impediment to the escape of bile from the liver. On the other hand, as in acute atrophy of the liver, the jaundice is usually associated with hæmorrhages, 'black vomit,' delirium, and the other symptoms of the typhoid state. There are good reasons also for believing that this typhoid condition is due to the same cause as in acute atrophy, viz., impaired or deranged metamorphoses in the blood and tissues, and retention in the system of those products of metamorphosis which ought to be eliminated by the kidneys. The urine in most cases is albuminous, and contains tube-casts, and is occasionally suppressed. Roche has found a deficiency of urea in the urine, but a large quantity of it in the blood; * Blair has detected a large amount of carbonate of ammonia in the blood, and also in

* Yellow Fever, Philadelphia, 1855.

the expired air;* while Lallemand describes the sweat as of a penetrating urinous odour.† The liver at first is enlarged from hyperæmia, but in the advanced stage of the disease it is pale and reduced in size, and the secreting cells are often loaded with oil.‡ The kidneys are also usually found to be large and congested in the early stage, but later in the disease the cortex is hypertrophied, and the secreting tubes gorged with granular epithelium. From what has been stated it seems but fair to conclude that the jaundice of yellow fever is only one of the results of that impairment or derangement of the metamorphoses taking place in the blood and tissues, of the existence of which there is such abundant proof.

b. Malarious Remittent and Intermittent Fevers.

The occurrence of jaundice in the malarious remittent and intermittent fevers of India, Algeria, and other countries where true yellow fever is believed to be unknown, has been repeatedly noted. Fifteen years ago I met with it in the malarious fevers of Burmah; || and Morehead, one of the latest and best writers on Indian diseases, observed jaundice in twenty-eight

* Report on Yellow Fever, by Daniel Blair, M.D., pp. 39, 40. Brit. and For. Med. Chir. Rev., April 1856.

† Ererichs, Op. cit., i. p. 183.

‡ Of thirteen fatal cases dissected by Louis at Gibraltar the consistence of the liver was diminished in seven. 'Its colour was altered in every case; sometimes it was of the colour of fresh butter, sometimes of a straw yellow, or of a clear coffee and milk colour, sometimes of a gum yellow, sometimes of an orange yellow.' Graves, Clin. Lect., 2nd. ed. i. p. 283.

|| Notes on the Climate and Diseases of Burmah, Ed. Med. and Surg. Journ., April 1855, p. 229.

out of one hundred and fourteen cases of remittent fever.* In Algeria jaundice has been sometimes noted in as many as seven-tenths of the cases of intermittent fever.† The jaundice in these cases arises in different ways. Sometimes it is associated with congestive enlargement of the liver, or with gastro-duodenal catarrh, more or less obstructing the flow of bile and causing deficiency of bile in the motions; in both these cases the general symptoms are often mild. But in other cases which are usually fatal, jaundice is found associated with a dry, brown tongue, drowsiness, delirium, tremors, subsultus, and other symptoms of the typhoid state, with petechiæ and hæmorrhages from the stomach and bowels, and with albuminous and bloody urine, which is sometimes completely suppressed.‡ Careful analyses of the blood and urine in these cases have still to be made, but there can be little doubt that the general condition is similar to, if not identical with, that of the typhoid state in yellow fever, in British typhus, and, in fact, in acute diseases generally.|| In these

* Clinical Researches on Disease in India, 2nd ed. 1860, p. 73.

† Boudin, *Traité des fièvres intermit.* Paris, 1842.

‡ Frerichs, *Op. cit.*, vol. i. p. 180.

|| In 1853 I found no albumen in the urine of persons suffering from remittent fever in Burmah. My observations, which, for the most part, were made early in the disease before the supervention of typhoid symptoms, have been quoted as establishing a distinction between malarious remittents and true yellow fever. The comparative frequency, however, of albuminuria in yellow fever is probably due to the fact that the typhoid state is much more common in this disease than in malarious remittents. When the typhoid state is developed in remittent fever, it would indeed be extraordinary, if it differed from the typhoid state of all other diseases, in the absence of albuminuria.

severe cases of remittent fever the bile-ducts have been found by Morehead and other observers perfectly patent and free from catarrhal inflammation, while the liver has been noted to be but slightly congested, and sometimes pale and in a state of fatty degeneration. The jaundice in these cases then, appears to result from a condition of the blood unfavourable to the metamorphosis of the absorbed bile.

c. Relapsing Fever.

Jaundice has been a frequent symptom in the relapsing fever of Great Britain and Ireland. Indeed, the frequency with which relapsing fever has been complicated with jaundice, and even with black vomit, has often caused it to be mistaken for true yellow fever. In 1826, Drs. Graves and Stokes published an account of the 'yellow fever' of Dublin, and the twenty-first chapter of the first volume of Graves' unrivalled 'Clinical Lectures' is entitled, 'Yellow Fever of the British Islands.' It is now generally admitted, that the cases described by these writers were instances of relapsing or famine fever, complicated with jaundice and cerebral symptoms, and their distinctness from true yellow fever was pointed out at the time by O'Brien.* The Scotch epidemic of 1843 was likewise regarded as closely allied to, if not identical with, yellow fever, by many of its most distinguished observers, and it was even fancied that the disease had been imported into

* Trans. Queen's Coll. of Phys. of Dublin, 1828, p. 532.

Glasgow by merchant-vessels from the West Indies, although in truth it had been prevailing in the east of Scotland for some time before it appeared in Glasgow.* There is, no doubt, a strong resemblance between the more severe forms of relapsing fever complicated with jaundice and typhoid symptoms, and tropical yellow fever, but we have here another illustration of the mistakes which, I have told you, are apt to result from founding analogies or differences between acute specific diseases, on symptoms alone, and of neglecting the circumstances under which they appear, or, in other words, their causes.

The frequency of jaundice in relapsing fever has been variously estimated, but on an average it may be said to have been noticed in one out of every five cases. The jaundice is independent of any obstruction to the escape of bile from the liver. In many cases the associated symptoms are mild, and the patients recover, and then the jaundice is probably the result of the congested condition of the liver, which is so common in relapsing fever. Yet most observers of relapsing fever have agreed in making jaundice a formidable symptom, and it has certainly been often accompanied by hæmorrhages, including 'black vomit,' a dry brown tongue, delirium, coma, subsultus, convulsions, and other cerebral symptoms, and at the same time it has not necessarily been associated with hepatic congestion, but often with a soft,

* Murchison, on the Continued Fevers of Great Britain, 1862, pp. 47, 364.

pale, and yellow condition of the liver.* As in true yellow fever, however, these serious symptoms are not due to the presence of bile in the blood, but the jaundice is only an outward and visible sign of important changes in the blood, interfering with the natural metamorphoses. The urine has been ascertained to be suppressed or diminished in quantity, and very deficient in urea, which has been found in abundance in the blood and in the cerebral fluid.†

d. Typhus Fever.

Very opposite statements have been made as to the occurrence of jaundice in true typhus fever. Sir W. Jenner states that he has never met with it, whereas, according to Frerichs, several epidemics of 'petechial typhus' have been characterized by the frequency of jaundice. It is probable that Frerichs has been misled by the frequency with which epidemics of typhus and relapsing fevers have prevailed together, and by the fact that in most instances the latter disease has been regarded as a mere variety of the former. At all events, in this country and in Ireland

* In two fatal cases of relapsing fever with jaundice and hæmorrhage and typhoid symptoms, recorded by Dr. Rose Cormack, the liver was found in one 'of the natural colour and consistence,' and in the other it was 'softer than natural,' and 'the section exhibited a dingy lightish colour.' 'Nat. Hist. Path., and Treatment of the Epidemic Fever at present prevailing in Edinburgh,' &c., 1843, Cases VII. and VIII. In the two typhoid cases reported by Dr. Graves, the appearance of the liver was perfectly natural. Clinical Lectures, 2nd ed. i. pp. 285, 288.

† See the evidence on this matter collected in my work on Fevers, p. 364.

jaundice is a very rare complication of true typhus. In 1843 Dr. Henderson referred to the occurrence of jaundice in typhus fever; * two cases are recorded by Frerichs,† and one in my work on the ‘Continued Fevers of Great Britain;’ Dr. Hudson of Dublin also speaks of jaundice as ‘a very rare complication in typhus.’‡ The rarity of jaundice in typhus, as well as the severity of the cases in which it occurs, may be judged of from what has been observed at the London Fever Hospital, since the publication of my work on Fevers. Out of 7,604 cases of true typhus admitted into the Hospital during the years 1862, 3, 4, and 5, jaundice was noted in only 16, or once in every 475 cases. Of the 16 cases, 12 were fatal, and deducting 2 cases where the jaundice did not occur until convalescence, and was evidently catarrhal, of the remaining 14 patients in whom jaundice coexisted with the typhus rash, 12 died. As in the specific diseases already mentioned, the jaundice is not due to any obstruction of the bile-duct; it is likewise independent of hepatic congestion. The hepatic tissue in the cases which I have had an opportunity of dissecting has been preternaturally pale and soft, and the secreting cells have contained a large quantity of oil, while both Frerichs and myself have found leucine and tyrosine in both the hepatic and renal tissue, and also in the urine.|| In

* Edin. Med. and Surg. Journ., 1844, vol. lxi. p. 220.

† Op. cit., i. pp. 168, 170.

‡ Lectures on the Study of Fever, 1867, p. 88.

|| Specimens of Leucine and Tyrosine from the urine of persons suf-

one of my cases (Case LXV.) also it was ascertained that, as in yellow atrophy (see p. 229), urea had almost disappeared from the urine. Excepting for the presence of jaundice, there is nothing very remarkable in these cases of typhus. Typhoid symptoms are always present in a prominent degree, and, as I have elsewhere endeavoured to show, these symptoms are probably due to an impaired elaboration and retention in the system of those products of blood and tissue metamorphosis, which ought to be eliminated by the kidneys. Convulsions, which may be regarded as the acme of the typhoid state, are now acknowledged to have a uræmic origin in typhus as well as in scarlatina, and I have placed on record cases of typhus,* without as well as with convulsions, in which urea has been found in the serum of the blood. When jaundice then occurs in typhus, it does not account for the other serious symptoms with which it is usually associated, nor does it in itself, probably, contribute in any way to the fatal event; it is merely one indication of an unusual impurity and derangement of the normal metamorphoses of the blood, as the result of which the absorbed bile is not transformed as in health, or even as in ordinary cases of typhus.

Occasionally jaundice in typhus admits of another explanation than that now offered. Its appearance

fering from typhus complicated with jaundice, were exhibited by me to the Pathological Society on Feb. 3, 1863. Path. Trans., vol. xiv. p. 269.

* See my work on Fevers, pp. 161, 166, 174.

in Case LXVII. was probably determined by the double pneumonia, and in Case LXVIII., where it occurred during convalescence, it seemed due to a condition approaching that of pyæmia, rather than to be a direct result of typhus.

e. Enteric or Pythogenic Fever.

I have met with jaundice in only two cases of enteric fever. In one (Case LXIX.) which recovered, it occurred during a relapse of the fever, and was probably due to catarrh of the bile-duct; in the other (Case LXXI.), it appeared on the fourteenth day, and was associated with albuminuria, and during convalescence with thrombus of the femoral veins; the albuminuria persisted, and the patient died within six months. In a third case communicated to me (Case LXX.), the jaundice came on towards the termination of a severe attack, and persisted through convalescence. A case is recorded by Andral where the jaundice was noted at the commencement of the third day, and where the patient died on the ninth day of pneumonia of the left lung.* Louis has recorded two fatal cases, one of which was associated with parotid bubo and secondary purulent deposits in the liver, and the other with erysipelas of the leg.† Sir W. Jenner never met with jaundice in enteric fever, but refers to a preparation from a fatal case

* Clinique Médicale, 3me éd. 1834, tom. i. p. 10.

† Recherches sur la Fièvre typhoïde, 2me éd. Paris, 1841, Obs. 17 and 26.

which occurred on the west coast of Africa. Two fatal cases are recorded by Frerichs.* In one the jaundice did not appear until the thirty-seventh day, when the patient appeared to be convalescent, and on the forty-first day the patient died with symptoms of pulmonary oedema; the urine was scanty, and after death the kidneys were found to be congested, while leucine and tyrosine were discovered in the hepatic tissue. In the other the jaundice appeared as early as the fifth day, and was accompanied by profuse epistaxis and violent delirium; death occurred on the eighth day before the commencement of ulceration in the ileum, and the liver was found to be in a state of acute yellow atrophy. Jaundice appears to be on the whole a rarer symptom in enteric fever than in typhus, and of the few cases where it occurs in some the jaundice is probably due to catarrh of the bile-duct. In others, however, its pathology appears to be the same as that of jaundice in the specific diseases already considered, or it arises from some impairment or derangement in the natural metamorphoses of the blood.

f. Scarlatina.

You will find in Dr. Graves's 'Clinical Lectures,' (vol. i. p. 453), two cases of scarlatina referred to which were complicated with jaundice and enlargement of the liver ascribed to 'hepatitis,' and that a chronic form of hepatitis is spoken of as a common

* Op. cit., vol. i. pp. 172, 215.

sequel of scarlet fever. Dr. G. Harley has also related a case of scarlet fever complicated with jaundice from what was believed to be congestion of the liver.*

From my own experience I am led to the conclusion that jaundice in scarlet fever is extremely rare. Out of about 2,000 cases that have come under my care, I have known it occur only in five. Three of the five cases were fatal. In two of the fatal cases an autopsy was performed; in one the liver was not congested in the slightest degree, but was pale and fatty (Case LXXIII.); in the other (Case LXXII.), it presented a nutmeg appearance, the margins of the lobules being pale and their centres full of blood; in both cases the bile-ducts were perfectly patent; the urine in both cases contained albumen, but it is to be regretted that no examination was made for leucine or tyrosine. It is very probable that when jaundice appears in scarlet fever, it may be sometimes due to hepatic congestion, or to catarrh of the bile-ducts; but in other cases, and these are the more fatal, it is evidently independent of congestion or of obstruction of the ducts, and is probably the result of serious derangements in the metamorphoses of the blood.

g. 'Epidemic Jaundice.'

Most writers on jaundice refer to its occasional occurrence in the epidemic form. You will find an account of several epidemics of jaundice in Frerichs's

* Pathology and Treatment of Jaundice, p. 93.

work on the Liver.* These epidemics have varied greatly in their fatality, and probably also in their nature. In some, not a single patient has died. This was the case in an epidemic at Chasselay, referred to by Frerichs, where the jaundice commenced with gastric catarrh, and the stools were always pale. A similar observation was made at Pavia in 1859. Of 1022 French troops stationed there, 71 were attacked with jaundice, but all recovered; the cases were characterized by pain in the epigastrium and hypochondria, and enlargement of the liver and spleen; and the epidemic was attributed to marsh miasmata, conjoined with unusual heat, fatigue, and intemperance.† On the other hand, in an epidemic which prevailed at Essen, in 1772, which attacked chiefly children, assumed an intermittent type, and was characterized by delirium and other nervous symptoms, a large proportion of the patients perished. In another epidemic which occurred in the island of Martinique, in 1858, the disease was extremely fatal among pregnant females; of 30 women attacked at St. Pierre during pregnancy, 20 aborted and died, death being preceded by delirium, coma, and other symptoms, closely resembling those of acute atrophy of the liver.‡ A few years ago a remarkable epidemic of jaundice occurred at Rotherham, where the condition of the drainage was notoriously bad. In the autumn of 1862 Rotherham

* Op. cit., i. p. 188.

† Medical Times and Gazette, June 8, 1861, p. 607.

‡ Brit. Med. Journ., Feb. 7, 1863.

was visited by a very fatal outbreak of enteric fever. This was followed by an epidemic of jaundice early in the following year, and in the month of February it was stated that not fewer than 150 persons were suffering from it, but that none who had passed through the fever in the previous autumn had been attacked.*

Most of these epidemics seem to have been due to some malarious poison; some perhaps may have resulted from a chill or other atmospheric influence (see *antea*, p. 133). The mechanism of the jaundice has probably varied with the severity of the epidemic. Sometimes it appears to have been occasioned by congestion of the liver or catarrh of the bile-ducts, but in others where it was accompanied by delirium and typhoid symptoms and was extremely fatal, and where the whole phenomena bore a close resemblance to those of acute or yellow atrophy of the liver, it has more probably been due to morbid conditions of the blood interfering with the normal metamorphoses.

2. *Animal Poisons.*

a. Pyæmia.

In a large proportion of cases of pyæmia, whether from external wounds or injuries, from parturition or from internal causes (see p. 150), there is jaundice of the skin, conjunctivæ, and urine. Many cases of this sort have come under my notice in the London Fever Hospital. The jaundice usually commences early in the disease, and continues to increase till

* Lancet, 1863, vol. i. pp. 222, 374.

death; but it is rarely intense, and sometimes it is so slight that it is apt to be overlooked. The bowels are usually relaxed and the evacuations contain plenty of bile. Occasionally, as I have explained to you in a former lecture (see p. 147), the liver is found to contain purulent deposits, but in most cases nothing can be detected in it to account for the jaundice. The organ is pale and anæmic, and the bile-ducts are patent and free from inflammation (see Case LXXVI.).* The urine, in addition to bile-pigment, often contains albumen or blood, indicating a condition of the kidneys unfitting them for eliminating the large quantity of urea which is manufactured in pyæmia in common with other pyrexial diseases. In most cases of pyæmia, the tongue after a time becomes dry and brown, and there are more or less stupor and delirium, and in fact all the phenomena of the typhoid state met with in typhus and in other diseases. The abnormal condition of the metamorphic processes going on in the blood and the accumulation in the blood of the products of metamorphosis which ought to be eliminated by the kidneys, to which this typhoid state is due, leads also to an impaired consumption of the bile which has been absorbed into the blood, and accounts for the jaundice.

* Virchow has lately maintained that the jaundice in pyæmia is catarrhal, and due to a plug of viscid mucus in the duodenal orifice of the duct (Virchow's Archiv. 1865, xxxii. Hft i.). According to Frerichs, however, 'the bile-ducts are open and usually pour out a little thin secretion,' and this coincides with my own experience. Moreover, the fact that the stools always contain bile and that the jaundice is in most cases slight, is opposed to this being the result of mechanical obstructions of the duct.

b. Snake-bites.

Since the days of Galen it has been known that the bites of snakes and vipers occasionally cause jaundice. The jaundice may be intense, and what is very remarkable is the rapidity with which it is sometimes developed. Speaking of cases of this sort, Dr. Mead long ago observed, ‘*intra non integram horam fit flavus, quasi ejus qui ictero laborat.*’*

Careful records of post-mortem examinations in cases where death has been due to snake-bites are still wanting, but it is clear that the jaundice is independent of any obstruction of the gall-duct, as the vomited matters and stools always contain bile. The very rapidity also with which the jaundice is developed is opposed to its immediate cause being congestion of the liver, and suggests the idea that it is the result of disordered innervation, whereby there is induced an abnormal condition of the metamorphic processes going on in the blood. The general symptoms resulting from snake-bites,—viz. : a quick small, irregular pulse, tendency to fainting, bilious vomiting, difficult breathing, cold perspiration, dulness of vision, injury of the mental faculties, and sometimes convulsions,† all point to serious derangement of the nervous system.

* *Tentamen de Vipera*, p. 36.

† *Christison on Poisons*, 1829, p. 470.

3. *Mineral Poisons.**a. Phosphorus.*

During the last few years numerous cases have been recorded, both in this country and on the Continent, of acute poisoning by phosphorus,* which are remarkable in this respect that in almost every instance jaundice has been one of the symptoms noticed. I do not refer here to those cases of chronic poisoning by phosphorus where there is necrosis of the jaw, so common in persons engaged in the manufacture of lucifer matches, but to cases where acute symptoms have followed one large dose of the poison. There has been much discussion as to what is the pathology of the jaundice in these cases. Virchow maintains that it is due to obstruction of the duodenal end of the bile-duct by thickening of the mucous membrane and a plug of mucus, and that although the stomach and duodenum have often been found to present no redness or obvious sign of inflammation, there is nevertheless a 'cloudy swelling' of the gastric glands and thickening of the whole membrane.† Dr. O. Wyss, however, has recently shown that when dogs with a biliary fistula were poisoned with phosphorus, jaundice was produced, which could not therefore be due to obstruc-

* An account of many of these cases will be found in the Year Books of the Sydenham Society, 1849, p. 445; 1860, p. 440; 1861, p. 409; 1862, p. 428; 1863, p. 404; 1864, p. 423: and Biennial Retrospect, 1865-6, p. 434. Two cases are also recorded in the Fiftieth volume of the 'Medico-Chirurgical Transactions,' by Drs. Habershon and Hillier.

† Archiv. f. path. Anat. u. Phys., xxxi. p. 399.

tion of the intestinal portion of the common bile-duct. After the appearance of jaundice much less bile escaped by the fistula, and that little was mixed with colourless mucus; sometimes mucus alone escaped.* This observation is interesting in connection with the fact that almost all the descriptions of the post-mortem appearances agree in stating that the liver is in an extreme state of fatty degeneration, and that as in acute atrophy, the secreting functions of the organ have been in a great measure abrogated. The appearance of the liver, in fact, has in many instances resembled very closely that of yellow atrophy. The symptoms moreover of acute phosphorus poisoning—drowsiness followed by violent delirium, convulsions and coma, vomiting, albuminous or bloody urine, and a fluid condition of the blood with petechiæ and hæmorrhages,† are utterly unlike those of catarrhal jaundice (see p. 132), and so closely resemble those of acute atrophy of the liver, that it has even been suggested that many of the recorded instances of acute atrophy have been really cases of phosphorus poisoning.‡ It seems probable therefore that the jaundice of phosphorus poisoning has a blood origin, and, like that of yellow fever and typhus, must be ascribed to an abnormal condition of the metamorphoses in the blood.

* Archiv. der Heilkunde, 1867, p. 419.

† It would be interesting to know if in cases of acute poisoning by phosphorus, the urine contains leucine or tyrosine, or if these substances are present in the tissue of the liver or kidneys.

‡ See for instance references in Syd. Soc. Year Book for 1862, pp. 428, 430, and for 1863, p. 404.

b. Mercury. c. Copper. d. Antimony.

The preparations of mercury, copper, antimony, and other irritant poisons have been known to cause jaundice, but only in exceptional cases. The mode of production of the jaundice has not been well ascertained, but the most probable explanation is that it is caused by the inflamed and swollen state of the mucous membrane blocking up the duodenal orifice of the bile-duct.

4. *Chloroform and Ether.*

Chloroform and ether, according to Frerichs,* occasionally cause jaundice, while several observers have found that under their influence sugar passes off in the urine. The concomitant symptoms of jaundice from these substances are little known, and the cases are extremely rare, for after considerable search I have been unable to find the records of any. Most probably the jaundice has a blood origin, but its precise mode of production has still to be determined.

5. *Acute Atrophy of the Liver.*

In a former lecture (see pp. 225, 238), I have shown you that the jaundice in that remarkable disease, acute or yellow atrophy of the liver, is independent of any obstruction of the bile-ducts, and that it is probably the result of some abnormal condition of the blood. The motions during life contain plenty of bile,

* Op. cit., vol. i. p. 160.

and after death the gall-ducts are found to be perfectly patent, while on the other hand all the phenomena of the disease approximate it to those maladies which are known to result from some poison, such as typhus, enteric fever, pyæmia, and phosphorus poisoning. I need only recall to your recollection the fact already adverted to in this lecture of leucine and tyrosine being found in the jaundice of typhus in common with that of acute atrophy, and the circumstance of the liver being found in a state of acute atrophy in a case of enteric fever complicated with jaundice. It becomes a question, indeed, whether the condition of the liver in acute atrophy be the cause of all the formidable symptoms with which it is associated, or whether it be not rather merely one of the consequences of some general disorder of the system, like that which is produced by many poisons. On a former occasion I told you that it had been repeatedly observed that several of the residents in the same house had been attacked with acute atrophy almost simultaneously (p. 233). There is good reason also for believing that some of the instances of 'epidemic jaundice' have been examples of acute atrophy. In the epidemic, for instance, which prevailed in the island of Martinique in 1858, the jaundice was accompanied by delirium, coma, and other symptoms of acute atrophy, and, as in acute atrophy, the disease was especially frequent and fatal in pregnant females, who aborted before death. It is a matter for investigation whether the anatomical changes which are so

notably present in the liver in cases of acute atrophy are really limited to that organ. Wagner, who is of opinion that many of the recorded instances of acute atrophy were probably cases of acute poisoning by phosphorus, on the ground of their complete clinical and pathological analogy with cases known to be of this nature, has drawn attention to the almost universal infiltration of every tissue of the body with oil in cases where death has been due to phosphorus, whereas this change had been previously recognized only in the liver. He found minute fat granules in the epithelium of the kidneys, in the parenchyma of the lungs, and in the muscular fibres of the voluntary muscles and of the heart. These observations have been confirmed by other investigators, and Bucquoy relates a case where even the brain was in a state of fatty degeneration.* One cannot fail to be struck with the analogy which, in this respect, cases of phosphorus poisoning bear to typhus, in which, as we have found, jaundice with leucine and tyrosine, is apt to be developed. It is now well known that a granular degeneration of the voluntary muscles of the heart and of the renal epithelium is among the most common post-mortem appearances in typhus, and probably in most diseases where death has been preceded for some time by the typhoid state. Speaking of the kidneys in acute atrophy of the liver, Frerichs observes:† ‘I have found the glandular epithelium

* *Union Médicale*, 1863, No. 81.

† *Op. cit.*, i. 227.

infiltrated with granules, and in most cases in a state of fatty degeneration, and the tissue itself flabby and shrivelled.' He also speaks of 'a flabby shrivelled character of the muscular tissue of the heart,' and states that, 'in some cases the cerebral substance has appeared softened,' although he expresses doubts whether this condition was the result of commencing putrefaction, or a product of disease. There seems reason then for believing that the condition of the liver in acute atrophy is only one of many similar changes taking place throughout the body, as the result of some blood-poison.

II. IMPAIRED OR DERANGED INNERVATION INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE OR INCREASING THE SECRETION.

That jaundice may have a nervous origin has long been known. There are numerous instances on record of its being produced by severe mental emotions, such as fits of anger, fear, shame, or great bodily suffering. Concussion of the brain has been known to have a like effect. Villeneuve relates the case of a young soldier, who, being insulted in public, drew his sword, and would have rushed upon the aggressor, but was restrained by the bystanders; in his vain efforts to wreak his vengeance, he became suddenly jaundiced; soon afterwards delirium set in, and he died in convulsions. He also quotes the case of a young abbé, who, owing to a sudden fright from a mad dog breaking its chain and rushing at him, uttered a loud cry, fell

down unconscious, and was taken up as yellow as saffron.* Mr. North witnessed a case in which an unmarried female, on its being accidentally disclosed that she had borne children, became in a very short time yellow; and a young medical friend of Sir Thomas Watson's had a severe attack of intense jaundice, which could be traced to nothing else than his great and needless anxiety about an approaching examination before the Censors' Board of the College of Physicians.† There are two circumstances worthy of note in these cases:—1. The rapidity with which the jaundice is developed, the skin and conjunctivæ become yellow almost in a moment, and even before the appearance of any bile-pigment in the urine; and, 2. that cerebral symptoms, such as delirium, coma, and convulsions often supervene upon the jaundice, and that the cases are then often fatal. These characters seem incompatible with the supposition that the jaundice can result from any mechanical obstruction of the bile-duct, or even from congestion of the liver, and make it more probable that it is caused by some derangement, through nervous influence, of the natural metamorphoses in the blood.

It is very probable, however, as Dr. Bence Jones has pointed out,‡ that jaundice has occasionally a nervous origin of another sort. The circulation and secretion of all glands are controlled by the nerves

* Dict. des Sciences, Méd., 1818, Art. Ictère, p. 420.

† Lect. on Pract. of Physic, 3rd ed. vol. ii. p. 557.

‡ St. George's Hospital Reports, 1866, vol. i. p. 193.

which supply them. Claude Bernard has shown that if the sympathetic filaments of the sublingual gland be tetanized, the blood in the gland becomes very dark, and the saliva scanty and concentrated ; but that if, on the contrary, the chorda tympani alone be tetanized, the blood in the gland presents an arterial hue, and the saliva is increased, though it contains a small proportion of solid matter. Similar results would no doubt be produced in the liver. Irritation of the sympathetic or paralysis of the branches of the pneumogastric nerve would probably contract the small blood-vessels and diminish the secretion of bile, while paralysis of the sympathetic, or irritation of the pneumogastric, would relax the capillaries and increase the rapidity of the circulation through the liver and the secretion of bile. Under these circumstances jaundice would be produced in the way which I shall explain to you presently when speaking of jaundice from congestion.

III. DEFICIENT OXYGENATION OF THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

Whatever interferes with a due supply of oxygen to the blood, interferes with those metamorphoses which in a healthy condition of the body are constantly taking place in it, and may thus impede or arrest the normal transformation of the absorbed bile and cause jaundice. It is deficient oxygenation which probably in a great measure accounts for many of the cases of true jaundice with bile in the stools (see *antea*, pp.

282, 329) met with in new-born infants. According to Dr. West, 'in the Dublin Lying-in-Hospital, where the children are defended by the most watchful care from the evils either of cold or of a vitiated atmosphere, the occurrence of infantile jaundice is rare: while in the Foundling-Hospital at Paris, jaundice is so common, that comparatively few infants escape it. Almost all the children at the Foundling-Hospital have been exposed to the action of cold while being brought to the institution, and suffer from the combined influence of cold and bad air while inmates of it—causes which interfere very seriously with the due performance of the functions of the skin and of the respiratory organs.' *

The jaundice which occasionally accompanies acute pneumonia in the adult may possibly have a similar origin. In the course of acute pneumonia the skin and conjunctivæ occasionally become jaundiced, and bile-pigment appears in the urine without any disappearance of bile from the motions. The pneumonia in these cases is far from being always in the lower lobe of the right lung, as some writers have stated. Of 19 cases observed by Drasche, the inflammation was in the right lung in 7—in 5 at the base, in 1 at the apex, and in 1 in the entire lung; in 8 the left lung alone was affected; and in 4 the pneumonia was double.† The jaundice in these cases is independent of any obstruction of the bile-duct, for, as a rule, there

* Lect. on Dis. of Infancy and Childhood, 5th ed. 1865, p. 602.

† *Österrh. Zeitsch. f. prakt. Heilk.*, 1860, No. 23.

is no lack of bile in the evacuations from the bowels. Various explanations have been offered as to its mode of production. It has been attributed to congestion of the liver from impeded circulation through the lungs, and possibly hepatic congestion may also be induced by nervous irritation reflected through the pneumogastric nerve from the lungs to the liver. Dr. Bence Jones has recently suggested that the jaundice in these cases is the result of an arrest of oxydation in the blood. These explanations, however, are not applicable in all cases. In some of the worst cases the proportion of the lungs implicated in the inflammation is comparatively slight, and the jaundice is associated with typhoid symptoms and albuminuria. The urine in these cases is often of a bright red colour, but, what is remarkable, it is said not always to present the ordinary reaction of bile-pigment with nitric acid. The absence of bile-pigment from the urine has accordingly been regarded as an unfavourable symptom in icteric pneumonia. Of 14 cases in which bile-pigment was found in the urine by Drasche, only 3 died; but of 5 where the urine contained no bile-pigment 2 died.* It is probable that in these cases the jaundice has a similar blood-origin to that of typhus, pyæmia, &c., which we have already considered.

Deficient oxygenation of the blood from inhaling a vitiated atmosphere in badly-ventilated or overcrowded apartments in many instances no doubt induces 'bilious headaches,' and functional derange-

* Oesterrh. Zeitsch. f. prakt. Heilk. 1860, No. 23.

ment of the liver, and may even conduce to the development of jaundice.

IV. EXCESSIVE SECRETION OF BILE, MORE OF WHICH IS ABSORBED THAN CAN UNDERGO THE NORMAL METAMORPHOSIS.

If we can suppose that in a particular individual the oxygenation and other processes of metamorphosis going on in the blood are just sufficient to transform the whole of the absorbed bile, it is not difficult to understand that in the event of the quantity of bile being increased, part of it might not be transformed and jaundice would be the result. Now this is probably what actually takes place in cases of congestion of the liver. The vessels of the liver are distended, and the diffusing surface of the walls is consequently increased, and more than the normal quantity of bile is taken up into the blood. In many cases of congestion of the liver the quantity of bile secreted is also increased. This then appears to be the pathology of the jaundice from congestion of the liver. There is no obstruction of the bile-ducts, unless there be concurrent inflammation of the duodenum or ducts (see p. 122), and sometimes, indeed, there is bilious diarrhœa. If the bowels be constipated, the jaundice from congestion of the liver will probably be increased, as the bile instead of being cleared away will accumulate in the biliary passages, and will be absorbed in all the larger quantity by the distended vessels. Mercury,

podophyllin, and other purgatives do good in these cases by sweeping away the bile as fast as it flows into the duodenum, and perhaps also by stimulating the gall-bladder and bile-ducts to contract through reflex action. As I have formerly told you (pp. 126, 309), there is no evidence that they stimulate the liver to increased secretion. If they did they would be injurious rather than otherwise in cases of jaundice from hepatic congestion.

The symptoms, varieties, and causes of hepatic congestion have been already considered in a former lecture (see p. 119).

V. UNDUE ABSORPTION OF BILE INTO THE BLOOD FROM HABITUAL OR PROTRACTED CONSTIPATION.

I have already explained to you one way in which jaundice may result from constipation, viz., from the pressure of *faeces* accumulated in the colon upon the bile-duct. Independently of causing pressure, however, it is very probable that a sluggish state of the bowels often contributes to the development of jaundice, partly by impeding the portal circulation and inducing congestion of the liver, and partly by causing an accumulation of bile in the biliary passages and duodenum, and thus favouring its absorption into the blood. It is under these circumstances that there is often developed the condition known as ‘biliousness from a torpid liver,’ where the patient suffers from languor, headache, furred tongue, flatulence and con-

stipation, a feeling of weight and oppression after meals, and not uncommonly hypochondriasis ; and, although these symptoms may last a long time without actual jaundice, this is liable to supervene at any time from irritating ingesta, or from other causes, increasing the congestion of the liver. The liver in these cases, so far from being ‘torpid,’ is perhaps secreting too much bile, while mercury and other purgatives do good, not as is generally supposed, by stimulating the liver to increased secretion, but by getting rid of a great portion of the bile as fast as it is thrown out, and thus preventing its absorption.

Treatment of Jaundice independent of Obstruction of the Bile-Duct.

The treatment of jaundice independent of obstruction of the bile-ducts must be regulated according to its cause.

1. In the jaundice from constipation, or in the state of ‘biliousness’ already adverted to which is short of actual jaundice, purgatives must be given in the first place, and of these the best are occasional doses of calomel, blue pill, or podophyllin, with salines, such as the sulphates of soda and magnesia, the citrate of magnesia, seidlitz powder, and the bitartrate of potash. Alkalies and their salts with the vegetable acids are also useful, partly in correcting acidity of the stomach, but mainly in carrying off by the kidneys the products of blood- and tissue- metamorphosis, the presence of which in the blood is the probable cause of the languor

and other symptoms from which the patient suffers. At the same time fermented liquors, wine, spices, fat, and all rich or indigestible articles of diet, which are calculated to irritate or congest the liver, must be forbidden. These are the measures most likely to relieve the jaundice or 'biliousness' resulting from constipation. But the great object of the practitioner in all such cases ought to be so to modify the patient's habits and diet, as to secure if possible a regular action of the bowels without the necessity of constantly having recourse to medicine. It is always well to enjoin regular exercise in the open air and the use of brown bread, or of such articles of diet as the individual knows from experience to have an aperient effect upon the bowels; and in cases where purgatives give only temporary relief, more permanent benefit may be expected from purgative mineral waters, such as those of Harrogate, Cheltenham or Leamington. When the constipation and its immediate effects have been relieved, but where the patient still suffers from weakness and symptoms of atonic dyspepsia, the mineral acids in conjunction with the vegetable bitters, such as quinine, gentian, or cascarilla, or with pepsine, may be expected to do good. Under these circumstances also chalybeates are sometimes useful, and especially in the form of mineral water, such as the waters of Kissingen, Pyrmont, Schwalbach, or Spa.

2. The treatment which is appropriate in cases of jaundice dependent upon congestion of the liver we have already considered, when treating of congestion

as one of the causes of enlargement of the organ (see p. 125). It is necessary to remember that some of the cases of jaundice occurring in the course of malarious and other fevers, or of pneumonia, or having a nervous origin, are due to hepatic congestion, and that then the treatment must be modified in conformity with the nature of the primary disease or cause.

3. In the jaundice arising from deficient oxygenation, the chief treatment must be the removal of the cause. In the jaundice of infants, which is independent of obstruction of the bile-ducts, the first thing to be done is to put the child in a wholesome atmosphere and to avoid exposing it to cold. The jaundice will then often disappear spontaneously without further treatment; but if it persist, a small dose of hydrargyrum cum creta, followed by castor oil, will often hasten its disappearance.

4. Lastly, in those terrible cases of jaundice associated with cerebral symptoms and the typhoid state, whether occurring in the course of malarious or infectious fevers, or in pyæmia, pneumonia, or the acute yellow atrophy of the liver, or having a purely nervous origin, it is not often that treatment is of much avail in averting a fatal result, but the measures most likely to do good are those which I have already recommended when speaking of the treatment of the acute yellow atrophy (see p. 234). Blisters* and sina-

* It is well to avoid cantharides for blistering purposes in these cases, when the urine contains albumen; but a blister can be readily produced even on the scalp, by applying to the skin for three or four

pisms to the nape and scalp, sinapisms to the feet, and remedies calculated to promote elimination by the skin, kidneys, or bowels, are sometimes of service. At the same time it will be necessary to support the patient's strength by diffusible stimulants and small quantities of alcohol.

The following cases illustrate the remarks now made on the subject of jaundice independent of any obstruction of the bile-ducts. Most of them occurred in my practice at the London Fever Hospital, where illustrations of the typhoid state, not only in the various specific fevers, but in many other diseases, are probably more numerous than in all the other hospitals of the metropolis put together.

The first three cases are examples of jaundice occurring in the course of typhus fever—an event, as I have told you, of extreme rarity. In two of the cases leucine and tyrosine were found in the urine; in the remaining case they were not looked for.

CASE LXV.—*Typhus complicated with Jaundice—Death by Coma—Leucine and Tyrosine, but scarcely any Urea, in Urine—Leucine and Tyrosine in Liver and Kidneys.*

Richard R—, aged 33, admitted into London Fever Hospital, August 26, 1862.

On admission, was too confused to give any account of himself;

minutes a piece of lint moistened with strong liquor ammoniæ, and covered with oiled silk. I have repeatedly produced a blister in this way with the best effect in cases of typhus complicated with albuminuria and cerebral symptoms.

pulse 120, feeble; tongue dry and brown along centre; skin warm and dry, with distinct typhus-rash, and a general yellowish tint. Was ordered beef-tea, milk, brandy (6 ounces), and a mixture containing sulphuric acid, sulphuric ether, and quinine.

The patient became weaker, and more unconscious. On the 28th, there was decided jaundice of the entire skin and of the conjunctivæ; the brandy was increased to 8 ounces.

August 29; pulse 120 and feeble; is scarcely conscious, and is inclined to be drowsy; pupils contracted. Decided jaundice of skin and conjunctivæ, and, at the same time, a well-marked petechial typhus-rash on chest and abdomen. Involuntary evacuations; tongue brown; motions light-coloured, but contain bile; no tenderness in hepatic region. Urine of a bilious colour, but does not yield the reaction of bile-acids; clear, acid, throws down no deposit, and contains no albumen; specific gravity, 1017. Six ounces of the urine were evaporated, and the residuum was found to contain abundance of globular masses of leucine and needle-shaped crystals of tyrosine, and also crystals of triple phosphate. When nitric acid was added to a drop of the urine, after concentration to one-twelfth of its volume, only a few small crystals of nitrate of urea could be discovered with the microscope. A blister was applied to the scalp; but the patient died comatose, at 3 P.M. on August 30.

Autopsy.—Deep jaundiced tint of entire surface. Heart and lungs healthy; blood, fluid and dark. Spleen, 7 ounces, very soft. Gall-bladder contained bile, which on squeezing flowed readily into the duodenum. Liver, 62 ounces, rather pale, and very friable, but lobules distinct; hepatic tissue contained numerous globular crystalline masses of leucine and tyrosine; secreting cells loaded with oil and bile-pigment. Kidneys enlarged, each weighing upwards of 7 ounces; surfaces smooth; cortex hypertrophied and containing crystalline bodies, similar to those found in the liver; uriniferous tubes gorged with epithelium. Intestines healthy, and their contents well coloured with bile.

CASE LXVI.—*Typhus Fever complicated with Jaundice.*

Henry B——, aged 42, was admitted into the London Fever Hospital, under my care, on September 24, 1862. He was in a state of delirium and stupor, and quite unable to give any account

of himself, but his body was covered with a petechial typhus eruption, the tongue was dry and brown, and the pulse 120 and feeble. There was also well-marked general jaundice of the skin and conjunctivæ, with bile-pigment and albumen in the urine. The abdomen was distended and tympanitic, but there was no tenderness or enlargement of the liver. The bowels were rather loose and the motions dark. The treatment consisted in nitro-muriatic acid, nitric ether, and taraxacum, beef-tea, milk, wine, and subsequently brandy.

The jaundice increased, and, although the pulse fell to 84, the patient became weaker, the urine had to be drawn off by catheter, and death took place in September 27.

Autopsy.—The intestines contained bile, and there was no obstruction of the bile-duct. The liver was pale and slightly fatty. The spleen was large and soft. The uriniferous tubes of the kidneys were gorged with granular epithelium.

CASE LXVII.—*Typhus Fever—Double Pleuropneumonia—Jaundice—Tyrosine in the Urine.*

James P—, aged 47, was admitted into the London Fever Hospital, under my care, on February 23, 1864, with the usual symptoms of a severe attack of typhus fever, the duration of which was doubtful. On admission, pulse 128 and feeble; distinct typhus eruption; tongue dry and brown; bowels confined; mind confused, and occasional delirium; signs of congestion at bases of both lungs. He was ordered mineral acids with ether, 6 ounces of brandy; milk, beef-tea, and an egg; and mustard and linseed poultices to the back of the chest.

On February 26 the patient had tremors and subsultus, and was lower, and a decided yellowness of the skin and conjunctivæ was noticed, but there was no enlargement or tenderness of the liver. The respirations were easy, and the lungs resonant on percussion. The urine threw down a copious deposit of lithates, both on February 26 and 27, and on the latter day the respirations were 40; there was dulness with tubular breathing over the lower third of both lungs; and the jaundice was more decided. The patient was ordered a mixture of ammonia, ether and senega, and the brandy was increased to 10 ounces.

On February 28, the pulse was 128, and the respirations 40;

the dulness of the lungs had extended. The jaundice was well marked, but there was plenty of bile in the motions. The urine was coloured with bile and gave a distinct reaction of bile-pigment with nitric acid, but contained no bile-acids (by Harley's test). Its specific gravity was 1018. On boiling it there was found to be a moderate amount of albumen (about $\frac{1}{16}$). On evaporating it down to a syrup, there were found numerous crystals of triple phosphate and yellowish-brown crystalline globules of tyrosine.

On February 29, the pulse was 140, and the respirations 60; the rash was fading, but the body was covered with profuse perspiration. He died at 8 P.M., on what was probably about the fourteenth day of his illness.

Autopsy.—Jaundiced tint of skin and conjunctivæ well marked. There was bile in the duodenum, and the bile-ducts were quite pervious. The liver was not at all congested, but was pale, soft, and very friable. The lobules were distinct, but the secreting cells in their pale rims were loaded with oil. The spleen was large and diffuent. The kidneys appeared normal, except that the cortices were pale, and the epithelium cells opaque and granular. No leucine or tyrosine could be detected in the hepatic tissue, or in the kidneys. The lower lobes and the lower part of the upper lobes of both lungs, were in a state of grey granular consolidation, and the pleural surfaces of the inflamed lungs were coated with a thin film of recent lymph.

In Case LXVII. the double pneumonia no doubt contributed to the development of the jaundice. In the following case (Case LXVIII.) the jaundice was a sequel of typhus, and coexisted with phlegmasia dolens and fatty degeneration of the liver, kidneys, and heart.

CASE LXVIII.—*Typhus Fever, followed by Phlegmasia Dolens, Jaundice and Death.*

Rosetta J——, aged 42, admitted into the London Fever Hospital, February 24, 1857. This patient had been ill for about eight or nine days before admission; and after she came under

observation in the hospital, her most prominent symptoms were:—pulse 120, extreme prostration; great restlessness and much low muttering delirium; involuntary stools and urine; well-marked typhus-rash; dry, brown tongue, and constipated bowels. The treatment consisted in wine, carbonate of ammonia, and castor oil to keep the bowels open.

About five or six days after admission, an improvement took place in the symptoms; and, by March 6, she had regained her strength to a considerable degree, her appetite was good, and pulse 80.

On March 9, which was about the 23rd day from the first commencement of the fever, and the 6th of convalescence, the patient felt ill again. Pulse 120, and small. Complained much of shooting pains in the left leg. Skin hot and dry. Some flushing of face. Tongue moist and very red.

The next day there was considerable swelling, and some tenderness of the left leg and thigh, but no hardness in the course of the femoral vein. The heart's action was heaving and tumultuous, but there was no bellows-murmur. Breathing short and rapid; no cerebral symptoms.

Blister over heart. Wine \mathfrak{zvi} . Saline efferv. mixture with tinct. hyoscy \mathfrak{zss} . every four hours. Left leg to be fomented and kept elevated.

No improvement took place, but at 4 A.M. of March 12 (the fourth day from first complaint of pains in the leg), the patient felt cold and chilly. There was a great increase of the prostration, and the pulse was imperceptible, although the heart's action continued tumultuous as before. Breathing very rapid. The mental faculties were unimpaired. The skin and conjunctivæ were of a marked yellow tint, and the face livid. Profuse sweating. No tenderness over liver, nor obvious increase of hepatic dulness.

Brandy and wine were freely administered, but the patient gradually sank and died towards evening.

Autopsy.—Cadaveric rigidity well marked. Distinct yellow tinge of skin on scalp, neck, and trunk. Thick layer of subcutaneous fat over the chest and abdomen. Copious sudamina over chest. Left leg swollen. Left ankle $8\frac{3}{4}$ inches in circumference, right $8\frac{1}{4}$; left calf 13 inches, right $11\frac{1}{2}$; left thigh 17 inches, right $14\frac{1}{2}$. Cerebral membranes moderately congested, and separated readily from brain. Subarachnoid fluid and fluid in ventricles of a decidedly yellow tint. Substance of brain tolerably firm;

red points numerous. Half an ounce of yellow serum in pericardial sac. Heart $8\frac{3}{4}$ ounces, valves normal; left cavities empty, and right almost empty. Walls of right ventricle very thin, and at apex composed almost entirely of fat. Substance of heart pale and soft, and, on microscopic examination, transverse striation indistinct, and fibres presented a granular aspect. Left femoral and iliac veins healthy, and contained no adherent clot. Each of lungs weighed 25 ounces; left, adherent throughout, and very emphysematous; lower lobes of both lungs much congested; no consolidation. Stomach and intestines healthy. Liver 52 ounces; capsule separated readily; substance of organ pale and very soft and friable, so that it broke down on removal; all trace of lobules had disappeared, the cut surface presenting a marrow-coloured pulpy appearance. On microscopic examination, many of the secreting cells could be seen loaded with oil, others appeared to be breaking up and disintegrating, and there was much free oil and granular matter. A small quantity of thick bile in gall-bladder; the bile-ducts quite pervious. Spleen 13 ounces, soft and pulpy. Kidneys enlarged; left, $7\frac{1}{2}$ ounces; right, 7 ounces: capsules separated readily; outer surface smooth; substance pale and flabby; cortical substance pale and granular, and rather increased in amount; uriniferous tubes gorged with oily epithelium.

The following are the only three instances of enteric fever complicated with jaundice of which I have notes. In the first (LXIX.) the jaundice occurred during a relapse of the fever, and was probably catarrhal; in the second (LXX.) it came on during the acme of the fever, and persisted during convalescence, in the third (LXXI.) it coexisted with phlegmasia dolens and albuminuria, as in the case of typhus already related (Case LXVIII. p. 411).

CASE LXIX.—*Enteric Fever followed by a Relapse with Jaundice.*

Mary A. C——, aged 43, was admitted into the London Fever Hospital, suffering from enteric fever; on February 9, 1863. She

had a dry, red, fissured tongue, with diarrhoea and rose-spots. She had been ill for nine days before admission, and on Feb. 25 she began to convalesce. She improved daily until March 5, when the febrile symptoms and diarrhoea returned, and on March 8, fresh rose-spots were observed. On March 11, the conjunctivæ and skin first became yellow, and on March 14, there was deep jaundice of the whole surface. The urine was dark green, deposited much lithates, and contained much bile-pigment, but no albumen, leucine, or tyrosine. The bowels, from the appearance of the jaundice, were rather confined, and the motions clay-coloured. The hepatic dulness measured 4 inches in the right mammary line; there was no tenderness below the right ribs. The tongue was dry, and there was great prostration, but no delirium. The patient was treated with nitro-muriatic acid and gentian, and linseed poultices over the abdomen. On March 16, the jaundice began to subside, and by the 21st, it had almost disappeared, and the patient was again convalescent.

The following case was communicated to me in a letter from the patient, who is a Fellow of the Royal College of Physicians.

CASE LXX.—*Enteric Fever complicated with Jaundice.*

One remark struck me in your book on Fevers. You refer to the extreme rarity of the complication of jaundice with typhoid. I myself was the subject of this affection in a very intense degree. This was in Paris in 1842. The jaundice came on suddenly about the acme of the fever. When the event was told to Rostan, who was seeing me, he said it was a ‘complication bien facheuse,’ and he did not expect that I would recover. This deep jaundice persisted for some time, even during convalescence, so that when I used to crawl into the Luxembourg Gardens I was known amongst the frequenters as the ‘Monsieur Jaune.’

CASE LXXI.—*Enteric Fever complicated with Jaundice and 'Phlebitis.'*

On December 12, 1863, I was requested by Mr. Edward Newton to see Mr. W——, a gentleman about 54 years of age, who, on September 30, had been taken ill with enteric fever, which presented the usual symptoms of diarrhœa, rose-spots, &c., till the fourteenth day, when he became jaundiced, and albumen appeared in the urine. During convalescence he got phlegmasia dolens of the left leg, with considerable tenderness along the course of the femoral vein; but after ten days this subsided, and the albumen disappeared from the urine. About a week before I saw him, he had driven out to the country, got out of the carriage and walked for five minutes. Within a few hours, the pain and swelling in the leg returned, and when I saw him on December 12, there was considerable pitting of the left leg, but only slight tenderness along the vein. The urine was turbid; sp. gr. 1013; it contained one-eighth of albumen and granular epithelial casts, but only a faint trace of bile-pigment, and no leucine or tyrosine. The impulse of the heart was barely perceptible, and the first sound was short and abrupt like the second.

The patient was treated with iron, quinine, a generous diet, and wine. At first he rallied somewhat, and on January 9, the swelling had almost left the legs, and there was no jaundice, and only a trace of albumen in the urine; but soon after this he became weaker, and he died at St. Leonards, in March, 1864. There was no post-mortem examination.

In the four cases which follow, jaundice appeared in the course of scarlet fever. In the first three, two of which were fatal, the symptoms indicated serious blood changes; in the fourth the jaundice was probably the result of simple congestion.

CASE LXXII.—*Scarlatina—Jaundice—Death by Coma.*

Samuel W——, aged 27, was admitted into the London Fever Hospital, under my care, on March 6, 1863, having been ill with

fever and sore throat for four or five days. On admission: pulse 120, weak. Copious, bright, punctated, scarlet eruption. Skin, especially of face, and conjunctivæ distinctly jaundiced. Tongue very red at edges, dry and brown along centre; throat sore; tonsils red and enlarged, not ulcerated; occasional hiccup; had vomiting and some purging before admission, no nasal discharge; mind clear. Ordered: carbonate of ammonia and chlorate of potash, 5 grains of each every four hours. Wine 6 ounces; beef-tea and milk.

During the night, the patient became quite unconscious, and his face was dusky and livid, and on the following morning, at 8.30 A.M., he died.

Autopsy.—Skin and white tissues deeply jaundiced. Bile-ducts patent. Liver presented a nutmeg appearance on section, the margins of the lobules being pale, and their central vessels containing blood. The oily matter in the secreting cells was much increased. The kidneys were large, the right weighing $6\frac{3}{4}$ ounces, and the left $8\frac{1}{4}$ ounces; capsules non-adherent, and surfaces smooth; cortices hypertrophied, five or six lines in thickness, dark-red and dripping with blood; uriniferous tubes gorged with finely granular epithelium. The urine from the bladder had a specific gravity of 1015, and contained a considerable amount of albumen and bile-pigment.

CASE LXXIII.—*Scarlatina—Jaundice—Sudden Death.*

Alfred C—, aged 19, was admitted into the London Fever Hospital, under my care, on December 4, 1862. His illness had commenced four days before with pains in the limbs and sore throat, followed by a scarlet rash which was now well out. Pulse 130; tongue moist, with white coat and red edges; bowels open; tonsils swollen and red, not ulcerated; no nasal discharge. Ordered: quinine (2 grains every four hours), milk and beef-tea.

December 5 (6th day). Pulse 136 and feeble. Had a restless night, wandering occasionally, but slept at intervals. Swallows and drinks well. Rash still out. Bowels open. Ordered: 4 ounces of wine.

December 6 (7th day). Was restless during the night, but slept at intervals. This morning the nurse observed his lips to

be slightly livid, and the face and conjunctivæ yellow, but otherwise the man seemed no worse. He asked the nurse to put his tea down, as it was too hot. Ten minutes later, the nurse saw him again, and found him unconscious, breathing quickly, and he died in five minutes, at 8.30 A.M.

Autopsy.—All the white tissues were tinged yellow, and the lungs were moderately congested posteriorly. The liver and bile-ducts presented nothing abnormal, except that the former was pale and slightly fatty. The kidneys were considerably congested, the uriniferous tubes were gorged with granular epithelium, and the urine in the bladder contained a small quantity of albumen.

CASE LXXIV.—*Scarlatina—Jaundice—Recovery.*

Emily S—, aged 18, was admitted into the Fever Hospital, April 5, 1864, having been ill one day with fever and sore throat. On admission, pulse 120; skin very hot; punctated scarlet rash of good colour and moderate intensity. Tongue red with white fur, and dry along centre; fauces red; tonsils large; no ulcer. No enlargement of glands in neck. Ordered a mixture with chlorate of potash and free chlorine, beef tea, &c.

April 6 (3rd day). Pulse 120, feeble. Bowels open three or four times since last night. Ordered, 4 ounces of wine and an egg.

April 7. During early part of last night was very restless and wandered, but slept fairly after an opiate draught. To-day, pulse 130; tongue dry; no ulcer of tonsils; answers correctly; intense scarlet rash on arms.

April 9 (6th day). Pulse 120, very feeble; rash fading; entire skin and conjunctivæ present a light jaundiced tint; no tenderness over liver; bowels open twice; motions pale yellow; tongue dry; desquamation commencing on face; slept badly, and mind wanders at times. Urine contains bile-pigment, but no albumen. Ordered 6 ounces of brandy.

April 11. Pulse 96; skin cooler; desquamation general; jaundice as before; three light-yellow stools; mind clearer; appetite returning, and girl feels better. Ordered quinine and custard pudding.

April 13. Copious desquamation. Jaundice fading.

April 17 (14th day). General health improving. To-day, for first time, skin is free from yellow tint. Bowels still slightly relaxed.

Was discharged well on April 29.

CASE LXXV.—*Scarlatina—Jaundice—Recovery.*

Frederick C—, aged 27, was admitted into the London Fever Hospital, December 17, 1861. Wife and child in hospital with well marked scarlatina. Was taken ill the day before with sore throat, rigors, and headache, and on admission, pulse 114; faint scarlet rash; tongue moist and furred, red at tip and edges; swallows with pain; tonsils large and red, but not ulcerated; bowels open. Was ordered an acid mixture, low diet, and beef tea.

December 19 (4th day). Face somewhat yellow; some tenderness over liver. Ordered two aperient pills.

December 20. Jaundice more decided; conjunctivæ yellow; tongue moist, clean, and red; throat less sore; bowels open three times; plenty of bile in motions; rash gone; pulse 84; and feels better.

December 21 (6th day). Jaundice more intense; urine contains much bile-pigment, but no albumen; slight tenderness on pressure over liver; bowels confined. Ordered a mixture containing nitrate of potash and sulphate of magnesia.

December 23. Pulse 66; less jaundice; no pain in hepatic region; bowels open several times.

From this date, the patient continued to improve, until his discharge on January 26, 1862. The jaundice disappeared in a few days. Desquamation was slight.

I have already brought under your notice several cases where jaundice resulted from pyæmia and where multiple abscesses were found in the liver (Cases XXXIII. to XXXVI., p. 155). In the following case the jaundice was also due to pyæmia, but there were no purulent deposits in the liver, and at the same time the bile-ducts were perfectly patent.

CASE LXXVI.—*Acute Necrosis of Cervical Vertebrae—Pyæmia—Jaundice.*

Elizabeth A—, aged 24, was admitted into the London Fever Hospital under my care on February 17, 1868. She had

been on the streets prior to her marriage, and had scars of buboes in the groins. Twelve days before admission she had been suddenly seized with acute pain in the back of her neck, which had never left her, prevented all motion of her head, and was accompanied by vomiting. She fancied that she had injured her neck while wrestling a week before her seizure, but her friends attached no importance to this as a cause of her illness.

On admission, the patient was suffering from symptoms of general fever, without any indication of local disease excepting that there were great pain and tenderness in the back of the neck and both shoulders, increased by movement, but without any obvious swelling or induration. The pulse was 108; tongue moist and red. Mind clear. No eruption on the skin, which was hot, with occasional perspirations; no rigors. A blister was applied to the back of the neck, and a mixture containing iodide of potassium and carbonate of ammonia (āā gr. iv.) and extract of belladonna (gr. $\frac{1}{3}$) was prescribed.

A few days after admission an obscure swelling could be felt down the neck on either side of the cervical vertebræ; there was a circumscribed pink flush on the left cheek; the tongue was dry and brown down the centre; and the patient complained of great burning and dryness of the throat. There was still occasional vomiting, and the pulse had risen to 126. On Feb. 28, the patient had several severe rigors; she had been slightly delirious in the night; there was diarrhœa with copious watery motions; there was a distinct pericardial friction over the heart, but no albumen in the urine. On March 2, there was slight but distinct jaundice of the skin and conjunctivæ, and bile-pigment in the urine, but there was no enlargement or tenderness of the liver, and the motions, which were still watery, contained plenty of bile; pulse 144; no return of rigors and no perspirations. A fluctuating swelling, the size of a small orange, was discovered a little behind the left ear, which on March 4 was opened, and discharged a quantity of thick pus. The patient was extremely prostrate, scarcely conscious, and very restless. The jaundice increased in intensity until her death, on March 5.

Autopsy.—The laminæ of all the cervical vertebræ excepting the first and two last were bare and bathed in pus. There was also pus in the spinal canal external to the theca and in the left lateral sinus. The left lung contained five or six small patches of lobular pneumonia passing into pus. The liver seemed healthy,

except that the secreting cells contained too much oil; it was not congested, and the bile-ducts were perfectly patent. There was no ulceration of the intestines.

Case LXXVII. is an example of jaundice occurring in the course of acute pleuro-pneumonia.

CASE LXXVII.—*Acute Pleuro-pneumonia complicated with Jaundice.*

On May 23, 1867, I was requested by Dr. W. H. Cook, of Hampstead, to see a clergyman between 50 and 60 years of age, of spare build and temperate habits, who had been long subject to spasmodic asthma, and who, five days before, had been seized with severe pain in the right hypochondrium, and febrile symptoms, followed by slight jaundice, cough, and high-coloured urine. On examination we discovered all the physical signs of pleuro-pneumonia of the lower half of the right lung—dulness, tubular breathing, fine crepitation, friction, and increased vocal resonance. There were also cough with considerable dyspnoea and tenacious rusty sputa, and acute pain in the right side, greatly aggravated by coughing or by taking a long inspiration. The pulse was 120 and the respirations 36. Along with these signs of pulmonary disease there was slight but decided jaundice of the skin and conjunctivæ, and the urine gave evidence of the presence of a small quantity of bile-pigment. The liver projected about half an inch below the ribs in the right mammary line, and there was a little tenderness over it on firm pressure. There was no deficiency of bile in the motions. The tongue was dry and brown, and there was great despondency as to the result of his illness. The patient was treated with carbonate of ammonia, nitric ether, stimulants, opiates, and other anodynes to relieve the pain and procure sleep, and mustard and linseed poultices to the side.

At first there was a marked improvement in both the physical signs and general symptoms, but early in June, pleurisy and then pneumonia made their appearance in the left lung, and the patient continued to sink until his death on June 14. To the last there was a slight ictteroid tinge of the skin and conjunctivæ, but the motions always contained plenty of bile. There was no post-mortem examination.

Case LXXVIII. appears to be a well-marked instance of jaundice from congestion of the liver. The fact of the jaundice following an attack of acute rheumatism, from Dr. Graves's observations (see *antea*, p. 291), led us to look for urticaria, but no such eruption appeared.

CASE LXXVIII.—*Jaundice from Congestion of the Liver.*

Jane G——, aged 30, was admitted into the Middlesex Hospital under my care on April 2, 1863. You will remember her as a patient in Seymour Ward from January 28 to March 9, suffering from acute rheumatism and pericarditis. After leaving the hospital she had complained of weakness and rheumatic pains, but she had been gradually getting better until March 26, when she was attacked with pain in the region of the liver, back and front, which on March 28 became much worse, and was followed on the next day by nausea, vomiting, and great flatulence, and on the 30th by jaundice. The vomiting only lasted for a day, but the jaundice and pain continued to increase.

On admission there was well-marked jaundice of the entire skin and conjunctivæ, and the patient complained greatly of pain in the epigastrium and right hypochondrium, stretching up to the right shoulder and down the back. The pain was greatly increased by pressure below the right ribs and also by lying on the left side, which the patient said always took away her breath. The area of hepatic dulness was increased in the right mammary line, measuring $5\frac{1}{4}$ inches, and extending quite an inch beyond the margin of the ribs. The urine was acid and dark, and contained a considerable amount of bile-pigment. The tongue was furred; there was no appetite; the bowels had been very freely moved by a dose of compound jalap powder taken on the day before admission, and the motions had contained plenty of bile. The pulse was 96 and the temperature 100° .

The whole hepatic region was dry-cupped and afterwards covered by linseed poultices; a draught of sulphate of magnesia and senna was ordered, with a diuretic mixture containing bitartrate of

potash and nitric ether. The diet was restricted to milk and beef tea.

On the following morning the catamenia appeared for the first time since the patient's confinement four months before; there had been frequent bilious motions, and the pain and tenderness in the region of the liver were greatly relieved.

On April 8, the patient was free from pain, and the jaundice was scarcely perceptible. A mixture with nitric acid and gentian was now prescribed and fish diet.

On April 13 the jaundice was quite gone; the hepatic dulness in the right mammary line was only four inches, and there was no tenderness below the right ribs. On the following day the patient was discharged.

Before concluding this lecture, I must say a few words on the diagnosis of the causes of jaundice, and more especially on the detection of bile-acids in the urine as an aid to diagnosis. In 1858 Kuhne announced that 'in jaundice resulting from closure of the ductus communis choledochus, the urine always contains biliary acid as well as bile-pigment;' but that under ordinary circumstances, when the ducts are free, 'the bile acids for the most part pass off by the fæces, and are not reabsorbed from the intestine.'* Dr. G. Harley, in his essay on jaundice, published in 1863, adopting the view that bile-acids are formed by the liver, while bile-pigment is pre-formed in the blood (see *antea*, p. 303), maintained that the detection of bile-acids in the urine was a means of distinguishing jaundice due to obstruction from that due to suppressed secretion. 'In jaundice from suppression,' Dr. Harley remarks, 'the liver does not secrete bile;

* Virchow's Archiv. Sept. 1858; and Translator's Preface to the English edition of Frerichs on the Liver, vol. i. pp. 14, 15, June 1860.

consequently no bile-acids being formed, none can enter the circulation, and they are therefore not to be detected in the urine ;' whereas in jaundice from obstruction, 'bile is secreted and absorbed into the blood, and the bile-acids not being all transformed in the circulation are eliminated by the kidneys and appear in the urine' (p. 60). Accordingly, it is argued that the presence or absence of bile-acids in the urine, in any case of jaundice, ought to decide whether this be due to obstruction or suppression ; and the readiest mode by which the bile-acids may be detected is said to be the following application of Pettenkofer's test : 'To a couple of drachms of the suspected urine add a small fragment of loaf sugar, and afterwards pour slowly into the test tube about a drachm of strong sulphuric acid. This should be done so as not to mix the two liquids. If biliary acids be present, there will be observed at the line of contact of the acid and urine—after standing for a few minutes—a deep purple hue. This result may be taken as a sure indication that the jaundice is due to obstructed bile-ducts.' On the other hand, 'a brown instead of a purple tint' is said to be equally indicative of suppression (p. 61).

If the views here announced should be confirmed, the pathology of jaundice would be greatly simplified, and physiological chemistry would have contributed an important aid to diagnosis. But after having bestowed considerable attention to the matter, it appears to me that both the theory and the practice based on it are open to objection.

1. For reasons already given (p. 303), the theory that jaundice, independent of obstruction of the bile-ducts, is due to *suppressed secretion* of bile, is itself a very improbable one.

2. Supposing the theory to be correct, bile-acids might be present in the urine unless the liver were *entirely* destroyed or the secretion *wholly* suppressed—events admitted to be of rare occurrence. In cases also of obstruction of long standing, the secreting tissue of the liver, as Dr. Harley admits, may be destroyed, and accordingly bile-acids might be absent from the urine, although the jaundice resulted in the first instance from obstruction. It follows that, however correct the theory, its practical application may mislead the physician in diagnosing the cause of jaundice.

3. The clinical evidence adduced in corroboration of the view that jaundice from obstruction is to be recognized by the presence of bile-acids in the urine is as yet insufficient. Neither Scherer,* a chemist of note, nor Frerichs has ever found bile-acids in the urine in any form of jaundice; and although minute quantities may possibly sometimes be detected by delicate tests such as that of Hoppe, after separating the urinary pigments, the necessity of which Kuhne admits, these methods are scarcely applicable to the ordinary purposes of diagnosis. Of the five cases of jaundice recorded by Dr. Harley,† in which the urine was tested for bile-acids, one was a case of jaundice

* Chemical Gazette, vol. ii. 1845, p. 208.

† Op. cit. pp. 27, 38, 74, 94, 111.

from obstruction of the common duct where abundant bile-acids were discovered on one occasion, but not a trace of them ten days afterwards; although seven weeks later, shortly before death, they reappeared in small quantity (p. 74). Another was a case of acute yellow atrophy of the liver, in which a decided reaction of bile-acids in the urine was obtained. This discovery might have been thought fatal to the diagnostic significance attributed to bile-acids in the urine, inasmuch as in acute yellow atrophy the bile-ducts are perfectly patent, and the disease has usually been regarded as a typical illustration of jaundice from 'suppression;' but Dr. Harley is of opinion that the presence of bile-acids in this case proves that in acute atrophy the suppression is complicated with reabsorption of bile (pp. 33, 38).

4. Since the promulgation of Dr. Harley's views, I have tested the urine in a large number of cases of jaundice, and have serious doubts as to Dr. Harley's modification of Pettenkofer's test, in which there is no provision for separating in the first place the urinary pigments, being a reliable indication of the presence of bile-acids in the urine. You will remember that on one occasion I applied the test to the urine of six patients under my care in the Middlesex Hospital. In three of the six cases a dark purple colour was developed at the line of junction of the sulphuric acid and the urine. One of the three cases was an example of jaundice from impacted gall-stone: in the other two cases, there was neither jaundice, nor any symptom

of disease of the liver, and yet when the three test-tubes were placed side by side, it was impossible to distinguish the colour in the first from that in the remaining two. Other observers, I believe, have arrived at similar results. I find also that Neubauer, in his excellent Monograph on the Urine,* states that certain of the pigments of the urine produce a dark purple-violet colour exactly resembling that from bile, when a large quantity of strong sulphuric acid is added to the urine. To separate the urinary pigments before testing for the bile-acids would obviously be too tedious and difficult a process for the ordinary purposes of diagnosis, even supposing that the presence or absence of the bile-acids threw more light on the cause of the jaundice than it probably would.

Although from these considerations I regret that I cannot recommend the test to which I have been adverting, as furnishing reliable information as to the cause of jaundice, the subject is one which you will do well to investigate for yourselves. Meanwhile, in forming a diagnosis you will sometimes be assisted by bearing in mind the remarks with which I bring this lecture to a close.

1. The chief indication of obstruction of the common bile-duct is furnished by the stools. When there is no obstruction of the duct, the stools almost invariably contain a certain quantity of bile; but when the duct is obstructed, no bile enters the bowel, and the

* A guide to the qualitative and quantitative Analysis of the Urine,
by Dr. C. Neubauer and Dr. J. Vogel, Syd. Soc. Transl. p. 48.

stools are clay-coloured. Two sources of fallacy must be remembered. First, the jaundice usually persists for a short time after the removal of the obstruction, and thus, as happens not unfrequently in the case of gall-stones, bilious motions may coexist with jaundice which has resulted from obstructed bile-ducts. Secondly, if the motions be thin or watery, they may appear to contain bile from the admixture of jaundiced urine.

2. A tumour corresponding to the region of the gall-bladder will favour the view that the jaundice is due to obstruction of the bile-duct (p. 142).

3. Jaundice which persists, and is yet slight, is most probably independent of obstruction of the bile-duct. Persistent jaundice from obstruction speedily becomes intense; but in reference to this you must remember, what I have so often insisted upon, that even when there is irremovable obstruction of the bile-duct, the intensity of the jaundice will vary from time to time according to the amount of bile secreted by the liver, and that in the advanced stage the jaundice may permanently fade in consequence of the destruction of the glandular tissue and the small quantity of bile which is secreted.

4. Jaundice appearing suddenly in a person whose previous health has been good is most probably the result of obstruction of the duct by a foreign body, or it has a nervous origin. In the former case it will be preceded or accompanied by biliary colic and vomiting, and the stools will be clay-coloured: in the

latter there will be a history of concussion or of some severe mental emotion, the motions will contain bile, and the jaundice will be often accompanied by delirium and other cerebral symptoms.

5. Jaundice coming on very slowly, but ultimately becoming intense, with complete disappearance of bile from the motions, is most probably the result of pressure on the bile-duct from without, or of the growth of some tumour in the interior of the duct (pp. 337-9).

6. Several attacks of temporary jaundice with distinct intermissions point to gall-stones if the patient be of adult or advanced life (p. 322) ; in early life, to catarrh of the duodenum or bile-ducts (p. 133).

7. Pain is present in some cases of jaundice, absent in others. There may be little or no pain in cases where the cause is a duodenal ulcer, a simple stricture of the duct, enlarged glands in the fissure of the liver, or the poison of some specific fever. It is well also to remember that in very rare cases a gall-stone has been known to obstruct the common duct and cause permanent jaundice, without ever having excited attacks of biliary colic. A pain coming on in severe paroxysms, and then subsiding, may result from : *a*, gall-stones (see p. 319) ; *b*, hydatids (p. 325) ; *c*, a duodenal ulcer (p. 334) ; and, *d*, an aneurism of the hepatic artery (p. 345). Pain, more or less constant, with tenderness on pressure below the right ribs, will indicate that the jaundice depends on : *a*, congestion of the liver (p. 121) ; *b*, catarrh of the bile-ducts

(p. 132); *c*, pyæmia with purulent deposits in the liver (p. 148); *d*, cancer of the liver (p. 190); *e*, acute atrophy of the liver (p. 227).

8. Jaundice concurring with great enlargement of the liver is most probably due to cancer of the liver (p. 190); but it may also arise from waxy liver, when the bile-duct is compressed by enlarged glands in the portal fissure (p. 26), or from multiple abscesses of the liver (pp. 147, 160).

9. The diagnosis of the cause of jaundice is often materially assisted by the coexistence with it of ascites. When permanent jaundice, with complete absence of bile from the motions, and ascites without dropsy elsewhere are present in the same case, you will rarely be wrong in inferring that the obstruction of the gall-duct, which causes the jaundice, and the obstruction of the portal vein, from which the ascites results, are due to a common cause. That cause cannot be a gall-stone. This will obstruct the bile-duct, but cannot obstruct the flow of blood in the portal vein, so as to produce ascites. The double obstruction is most likely to be caused by pressure from without upon the gall-duct and portal vein, where they lie side by side in the fissure of the liver, by enlarged lymphatic glands, by a tumour in the head of the pancreas, or by cancerous nodules projecting from the surface of the liver itself. It is quite possible, however, for these lesions to cause jaundice, without ascites. In the advanced stage of cirrhosis it is also not uncommon for jaundice to coexist with ascites;

but then the liver is small, the jaundice is slight—little more than sallowness, and, what is more important, the colour of the motions proves that bile-pigment is still secreted, and finds its way into the bowel. There is not a complete absence of bile-pigment from the excrement.

10. In a large proportion of cases of jaundice the pulse is unusually slow and the temperature is not increased. When jaundice is accompanied by febrile symptoms, the probable causes are: *a*, inflammation of the bile-ducts (p. 132); *b*, some specific fever (p. 378); or *c*, pyæmia (pp. 148, 390).

11. Delirium, stupor, and other cerebral symptoms concurring with jaundice suggest: *a*, acute atrophy of the liver (pp. 228, 395); *b*, poisoning by phosphorus (p. 393); *c*, some specific fever or other blood-poison (p. 378); *d*, nervous shock (p. 398); or *e*, pneumonia (p. 401). In all these cases the symptoms are those of an acute illness, the stools contain bile, and the urine often contains leucine and tyrosine and is deficient in urea. Similar symptoms, however, may also supervene in cases of protracted jaundice from obstruction of the bile-duct, in which the stools contain no bile (p. 296).

12. In diagnosing the cause of jaundice it is always important to keep in view the condition of the patient prior to its appearance. In the case of jaundice from gall-stones or nervous shock the patient may have been in excellent health previously. In catarrhal jaundice the attack is preceded for a week or ten days by febrile symptoms with vomiting or diarrhœa

(p. 132). Great emaciation with vomiting after food prior to the jaundice ought to suggest cancer of the pancreas, duodenum, or pylorus (pp. 337, 342), and pain two or three hours after a meal with hæmatemesis will point to a duodenal ulcer (p. 333). Jaundice occurring in the course of specific fevers or pyæmia will be preceded by the symptoms characteristic of these disorders. Jaundice in the early stage of pregnancy may be due to congestion of the liver from suppression of the catamenia (p. 125); in the more advanced stages it may arise from pressure of the enlarged uterus upon the bile-duct (p. 348), or from acute atrophy (p. 232). Lastly, true jaundice in the newborn child may result from the inhalation of a vitiated atmosphere (p. 401), from plugging of the bile-duct by inspissated bile or gall-stones* (p. 323), or from congenital closure or deficiency of the duct (p. 329).

* An instance of complete obstruction by a concretion of the pancreatic and biliary ducts, in an infant 25 days old, is given by Fauconneau-Dufresne, *La Bile et ses Maladies*, *Mém. de l'Acad. Roy. des Sc.* tom. xiii. p. 264.

LECTURE XI.

FLUID IN THE PERITONEUM.

ITS SIGNS—THE CONDITIONS WHICH SIMULATE IT, AND HOW TO DISTINGUISH THEM: 1. OVARIAN CYST; 2. HYDATID CYST; 3. RENAL CYST; 4. DISTENDED URINARY BLADDER; 5. PREGNANT UTERUS—CAUSES OF FLUID IN PERITONEUM: I. ACUTE PERITONITIS; II. CHRONIC PERITONITIS; III. CANCER; IV. COLLOID; V. SIMPLE DROPSY—1. FROM DISEASE OF KIDNEYS; 2. FROM DISEASE OF HEART OR LUNGS; 3. FROM PORTAL OBSTRUCTION.

GENTLEMEN,—Pursuing a similar course to that adopted when I was lecturing on enlargements of the liver and jaundice, I purpose to-day laying before you the various causes of fluid in the peritoneum, or ascites, and the means of distinguishing them, more especially in reference to diseases of the liver, of which ascites is so common a symptom.

The signs of fluid in the abdominal cavity are as follows:—

1. There is enlargement or swelling of the abdomen.

2. A dull sound is elicited on percussion over the seat of fluid. It is very common for persons in middle or advanced life to consult a medical man in the belief that they have got dropsy, the swelling being nothing more than an accumulation of gas in the bowels,

aided perhaps by an increase of the subcutaneous fat. The nature of the case will be at once revealed by percussion, which will give forth a clear sound over a tympanitic bowel.

3. A peculiar thrill or sense of fluctuation on percussion. This is elicited by laying the left hand flat on the side of the abdomen and then tapping abruptly, but gently, on the other side with the fingers of the right hand. This thrill is always most decided when the quantity of liquid accumulated is great, and when the abdominal wall is thin and tense; but even a few ounces may be detected by skilful hands. In this case, however, you must not expect to get the thrill propagated from one side of the abdomen to the other, but you must apply the fingers of the left hand over the upper margin of the part that is dull on percussion, and tap on the dull part a few inches below with the fingers of the right hand.

4. Pressure will sometimes give unmistakable evidence of fluid in the abdomen. If pressure be made with the tips of the fingers suddenly and perpendicularly to the surface, you will frequently experience a sensation of the displacement of liquid and of your fingers coming in contact with some solid body, such as an enlarged liver or spleen, or a tumour.

5. When the quantity of fluid is great, it will interfere with the proper action of the diaphragm and abdominal muscles, and cause more or less dyspnoea and thoracic breathing.

6. In cases also where there is a large accumulation

of liquid, the patient's head is often thrown back in standing or walking, to balance the body. The same gait is constantly observed in the advanced stage of pregnancy.

7. Mere accumulation of liquid in the abdomen, by compressing the renal and iliac veins, may give rise to albuminuria and anasarca of the legs, but these are important characters to which we shall return presently.

CONDITIONS SIMULATING FLUID IN THE PERITONEUM.

When the characters now enumerated are present, you may be perfectly sure that you have to deal with a collection of fluid in the abdomen, but they are not sufficient to enable you to say whether the fluid is in the peritoneal cavity or not, and this is the next point which you must always proceed to determine. Fluid in the peritoneum is most readily simulated by: 1, an ovarian cyst; 2, an hydatid tumour; 3, a large cyst attached to the kidney; 4, a distended urinary bladder; or, 5, a pregnant uterus: and in practice it is very necessary that you should avoid confounding with it any of these conditions.

1. *An Ovarian Cyst.*

From its great frequency this is the condition most apt to be mistaken for ascites. As long as an ovarian tumour is small, its outline can be felt through the abdominal parietes, and the diagnosis is easy. The difficulty arises when the cyst is very large and ap-

pears to fill the abdomen.* Even then, however, it is readily distinguished from ascites by the following characters, most of which also serve to distinguish ascites from other, as well as ovarian, cysts in the abdomen containing fluid :

1. In ascites the fluid, being free to move about among the bowels, always gravitates, and the intestines containing gas float on the surface, whatever be the position of the patient. Consequently, when the patient lies on his back, there will be dulness on percussion in the flanks and a clear tympanitic circular space, of greater or less extent, around the umbilicus, generally more above the umbilicus than below it, inasmuch as patients lie mostly with the shoulders more elevated than the pelvis. When the patient lies on his right side this clear space will shift to the left ; and when he lies on the left side, it will shift to the right, and in any situation, the dulness on percussion will often disappear at its margin on deep pressure. But in ovarian dropsy the cyst ascends in front of the intestines, which are prevented coming in front of it by the mesentery, and which are pressed back by the tumour against the spine. Accordingly, if there be any tympanitic resonance, it will be in one or both flanks, or in the epigastrium, and the umbilical region will be dull, while the relative position of the clear

* These remarks apply only to those cases where the tumour is entirely or mainly composed of one cyst. Multilocular ovarian cysts are readily distinguished from ascites by their uneven surface, greater hardness and resistance on pressure, and by the comparatively obscure fluctuation.

and dull spaces will not vary with the posture of the patient. These important differences between ascites and ovarian dropsy are illustrated in the annexed diagrams (figs. 24 and 25).

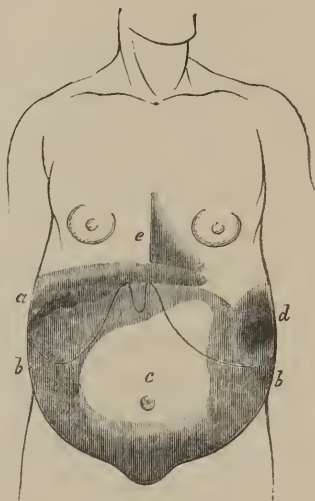


Fig. 24 illustrates the percussion-sounds over the abdomen in a case of ascites from cirrhosis of the liver.

a. Dulness of contracted liver. *b.* Fluid in peritoneum causing bulging of the flanks. *c.* Tympanic intestines. *d.* Enlarged spleen. *e.* Heart.

2. In ascites the swelling of the abdomen is uniform and symmetrical from the first, and when the patient lies on his back, the weight of the fluid causes a bulging on either side and gives an appearance of increased breadth to the trunk. But in ovarian disease the swelling commences in one flank, and for a long time is more on one side of the abdomen than the other; when large, also, although

it may appear to fill one side of the abdomen as much as the other, it bulges forwards rather than laterally.

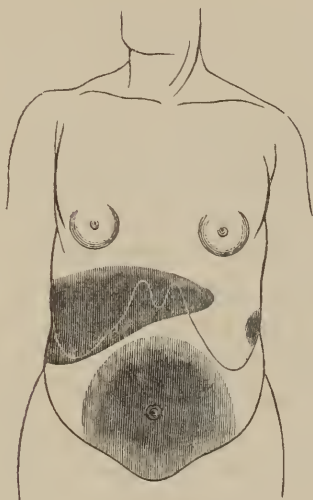


Fig. 25 illustrates the percussion-sounds over the abdomen in a case of tumour of the left ovary. The dullness of the tumour occupies the centre of the abdomen, which on either side is tympanitic.

3. In ascites the distances between the umbilicus and the pubes and sternum maintain their normal ratio, the navel being about an inch nearer to the pubes than to the sternum ; but in the case of ovarian tumour this ratio is often reversed. In the latter also, but never in ascites, the distance between the umbilicus and the crest of the ilium may differ on the two sides, being greater on the side from which the tumour has sprung. In ascites, when the patient is recumbent, the greatest girth of the abdomen is

usually at the umbilicus; but in ovarian tumour it is more commonly an inch or two below this.

4. In both ascites and ovarian tumour there is often obliteration of the umbilicus, but in simple ovarian tumour there is never any protuberance of the umbilicus, as is common in ascites. This is sometimes a sign of some importance as indicating the supervention of ascites on ovarian tumour.

5. There is sometimes a difference between ascites and ovarian dropsy in the fluid drawn off by tapping. In ascites it is either a clear, straw-coloured water, having a specific gravity of about 1012, and containing a large quantity of albumen, or, if there has been any inflammation of the peritoneum, the fluid may be turbid and contain flakes of lymph; whereas the fluid from an ovarian cyst, although often thin and almost colourless like that of ascites, is sometimes of a glutinous consistence and of a brownish or chocolate colour from the admixture of blood.

By attention to these rules, you will seldom have much difficulty in distinguishing between ascites and ovarian dropsy. There are, however, certain sources of fallacy in the diagnosis which it is necessary to keep in view.

a. When the quantity of fluid in the peritoneum is very great, the mesentery may not be broad enough to allow the intestines to float to the surface, and consequently there may nowhere be any tympanitic sound elicited on percussion of the abdomen.

b. The intestines in ascites may be prevented float-

ing by old adhesions or a diseased omentum binding them down to the spine. In cases, for instance, of sub-acute or chronic peritonitis, the intestines may be matted together and bound down to the back of the abdomen by old adhesions, while fluid collects between them and the abdominal wall in front (see Case LXXXII.).

c. In very rare cases an ovarian tumour has a coil of bowel stretched over it, which may yield a tympanitic percussion-sound.

d. An ovarian cyst may contain air as well as fluid, owing to decomposition of its contents after tapping, or to its having effected a communication with some portion of the bowel, as in Case LXXIX. Under these circumstances you may have an ovarian cyst with tympanitic percussion sound at the umbilicus.

e. Occasionally there is a concurrence in the same person of ascites and ovarian tumour. In this case, when the patient is supine, there is dulness on percussion at the umbilicus as well as in the flanks; but if he lie on the side from which the ovarian tumour springs, the percussion over the opposite side may be tympanitic. By pressing suddenly also with the points of the finger on the abdomen, in the manner already described, you may experience a sensation of displacing fluid, and at the same time of impinging upon an elastic tumour.

2. *An Hydatid Tumour.*

It is not often that an hydatid tumour fills the

abdomen to such an extent as to be readily mistaken for ascites, but occasionally a mistake of this sort is apt to arise. You will remember that in a former lecture (Case XIV., p. 102) I related to you the case of a girl of 15, in whom an hydatid springing from the liver caused such an enlargement of the abdomen as seriously to embarrass the function of respiration and threaten death from asphyxia—which was only temporarily averted by drawing off 248 ounces of fluid by paracentesis, and where the real nature of the case was only disclosed on post-mortem examination. Such a case, however, would be distinguished from ascites by the following characters :

1. By the swelling being in the first instance unsymmetrical, or confined to one portion of the abdomen, generally the hepatic region, before it becomes general. Most commonly an hydatid cyst commences in the hepatic region and grows downwards, but the possibility of its originating in the pelvis, and growing upwards, must be kept in view.*

2. By the tympanitic portion of the abdomen not always being most elevated whatever posture the patient assumes. In the case already referred to there was tympanitic percussion in both flanks, while

* See, for instance, a case recorded by Dr. Habershon in the *Pathological Transactions* (vol. xi. p. 155), where the tumour reached as high as the umbilicus and exactly resembled a distended urinary bladder; another reported by Mr. Bryant, where it reached as high as the scrobiculus cordis (*Path. Trans.* vol. xvii. p. 278); and a third reported in my paper on hydatid tumours in the *Edin. Med. Journ.* for Dec. 1865, Case X. In most of these cases the tumour interferes sooner or later with micturition.

the anterior portion of the abdomen was dull and fluctuating.

3. By the fluid drawn off being clear and limpid, strongly impregnated with common salt, but devoid of albumen or urea (see p. 61).

In those rare cases already referred to (pp. 56, 110) where ascites coexists with hydatid tumour, the difficulty of diagnosis will be considerable.

3. *A Renal Cyst.*

A cyst attached to the kidney occasionally acquires such dimensions as to cause great enlargement of the abdomen. Not long ago you had an opportunity of seeing a case of this sort (see Case XXIII., p. 115) where a cyst of the right kidney contained upwards of 200 ounces of fluid. However large such a cyst may be it is always easily distinguished from ascites :

1. By the signs of fluid being limited to one side of the abdomen, and by the tympanitic intestines being pushed over to the other, in whatever posture the patient lies.

2. By there being often a history of some injury to the kidney years before.

3. By there being hæmaturia or albuminuria, or some symptoms of urinary irritation. It is important, however, to remember that enormous renal cysts may exist independently of any urinary symptoms (see p. 115).

4. The fluid obtained by tapping may contain

urea, and on adding nitric acid to the alcoholic extract crystals of nitrate of urea would be produced. Mr. Stanley has recorded two cases of this sort where the fluid of the cyst contained urea.* This character, however, although of great value in pointing to the origin of a circumscribed cyst in the abdomen, would not distinguish the case from ascites, for in dropsy from diseased kidneys, the fluid that collects in the peritoneum and elsewhere has been often shown to contain urea in large quantity. Moreover, it is still a matter for investigation whether the fluid in all these renal cysts, and especially in those where there is no communication between the cyst and the pelvis of the kidney, contains urea (see p. 115).†

4. *A distended Urinary Bladder.*

You may think it very improbable that a distended urinary bladder could ever be taken for ascites, or indeed for anything else than a distended bladder, but such a mistake has more than once been committed, and there can be no doubt that the bladder in rare cases becomes so enormously distended as to fill a great part of the abdomen, and simulate ascites or cystic tumours. There is the authority of Sir Everard Home for the fact that John Hunter once actually tapped a distended bladder in the belief that

* An account of two cases of rupture of the ureter or pelvis of the kidney. *Medico-Chirurgical Transactions*, 1844, vol. xxvii. p. 1.

† Dr. Cooper Rose and Mr. Spencer Wells have recorded cases in which there was no urea.—*Lancet*, May 30, 1868.

the disorder was ascites,* and I show you here a bottle of urine obtained by paracentesis from a patient who was believed by one of the most experienced surgeons of the present day to be suffering from a large hydatid tumour of the abdomen. You may judge of the size of the bladder in this case when I tell you that 480 ounces of fluid were drawn off by a small trocar introduced midway between the umbilicus and the sternum. The case is one of such importance in diagnosis that I shall not be wasting your time in relating it to you in detail (see Case LXXX.). I may refer also to a case which occurred in my own practice a few years ago, where a sacculus of the bladder, in a man aged 68, became so enormously dilated as to form a large tumour in the right iliac region, which compressed the femoral vein and caused thrombosis of this vessel with painful swelling of the leg.† Cases of distended bladder will be distinguished from ascites:

1. By there being dulness at the umbilicus and displacement of the bowels upwards and laterally.

2. By there being in the earlier stages a central circumscribed tumour containing fluid, and growing upwards from the pubes.

3. By there having been in most cases at some

* Lect. on Practice of Physic, by Sir Thomas Watson, 3rd edit. vol. ii. p. 380.

† In this case also the patient passed normal urine freely and seemed to have no urinary symptoms. The cyst was tapped midway between the pubes and the spine of the ilium, and twelve ounces of urine drawn off, and the operation was followed by no bad consequence. The case is fully reported in the Pathological Transactions (vol. xiv. p. 133).

former period symptoms of retention or of some urinary disturbance, even though such symptoms may be absent subsequently when the patient first comes under notice,

4. Cases of this sort are most common in old men with large prostates, where an ovarian cyst, which most closely simulates the characters of a distended bladder, may be excluded from the diagnosis, and whenever from the circumstances any doubts exist as to the enlargement being due to the bladder, they may be solved by introducing a long prostatic catheter and pushing it well up. With an ordinary catheter only a few ounces of urine may be obtained, and no impression may be made on the tumour, for, as was long ago shown by Deschamps,* the bladder in these cases bends over upon itself, so that the greater part of the viscus is shut off from the neck, which is also distended so as to hold a few ounces of the urine.

5. *A Pregnant Uterus.*

Although from the time of Queen Mary there have been many celebrated instances of abdominal dropsy being mistaken for pregnancy, and *vice versâ*, it would be an unjustifiable error for a medical man at the present day to mistake a solid enlargement of the uterus, accompanied by all the constitutional symptoms and local signs of the pregnant state, for dropsy of the peritoneum.

* *Traité de l'Opération de la Taille*, tom. i. p. 224. This is a source of fallacy in diagnosis on which, more than twenty years ago, Mr. Syme was in the habit of strongly insisting in his Clinical Lectures.

CAUSES OF FLUID IN THE PERITONEUM.

But supposing you have decided that there is really fluid in the peritoneal cavity, the next point to determine is its source or cause. It may be due to: I. Acute Peritonitis; II. Chronic Peritonitis; III. Cancer of the Peritoneum; IV. Colloid disease of the Peritoneum; or V. Simple Dropsy.

I. *Acute Peritonitis* is distinguished by:

1. Its rapid course.
2. The symptoms of abdominal inflammation—pyrexia, with small rapid pulse, shrunk features, clammy sweats, and great tendency to collapse; vomiting; acute pain and tenderness of the abdomen; thoracic breathing; and legs drawn up.

3. The quantity of fluid thrown out into the peritoneum is usually small, and is often insufficient to produce a distinct sense of thrill or fluctuation.

II. *Chronic Peritonitis* is in most cases tubercular, and then the tendency is to the formation of firm adhesions of the abdominal viscera to one another and between them and the abdominal wall, without any accumulation of fluid. The abdomen in these cases is usually retracted. Occasionally, it is true, circumscribed collections of pus form between adjacent coils of bowel (from tubercular ulcers of the mucous coat ending in perforation, or from mere softening of tubercular matter), and acquire such a size as to cause a bulging of the abdominal wall, with signs of fluid (see Case LXXXIV.). But there is such a thing also as chronic peritonitis independent of tubercle, where the

intestines become matted together and tied down to the spine, and where fluid accumulates in considerable quantity between them and the abdominal wall in front (see Case LXXXII.). These cases are distinguished as follows :

1. There is symmetrical and rarely very great enlargement of the abdomen.

2. There is distinct fluctuation, but nowhere tympanitic percussion-sound, except perhaps at the epigastrium.

3. There is in most cases slight fever, with pain and tenderness of abdomen and thoracic breathing—the symptoms in fact of acute peritonitis in a very modified form. Occasionally the symptoms at the commencement are those of acute peritonitis.

4. The diagnosis may be assisted by there being an absence of other causes of fluid in the peritoneum, such as disease of the heart, liver, or kidneys.

III. *Cancer of the Peritoneum.*—It is not often that the peritoneum is the seat of cancer, but cases are every now and then met with, where cancer commences in, and at death is still limited to, the peritoneum. One peculiarity of these cases is that there always is more or less peritonitis, with the effusion usually of a large quantity of fluid into the peritoneal cavity, often necessitating paracentesis to avert asphyxia, and in this respect presenting a striking contrast to tubercular peritonitis. Four cases answering to this description have come under my notice. One was the case of Jane O—, aged 51, whose

symptoms I shall presently describe to you in detail (Case LXXXIII.). The second was the case of a rickety female aged 78, who died in this hospital on October 10, 1860, whose abdomen was enormously distended with many quarts of flaky fluid, and the intestines studded on their peritoneal surface with numerous small nodules of cancer of about the size of split peas. Three months before death she had been attacked with vomiting and abdominal pain, accompanied by emaciation, followed after six weeks by ascites, rapidly increasing till death. The third was the case of a man aged 39, who died in this hospital on Feb. 19, 1861, and in whose body I found appearances precisely similar to those in the second case. His illness had commenced $2\frac{1}{2}$ months prior to death, with pain and tenderness of the abdomen, followed by rapidly increasing ascites. The fourth was that of a bargeman, aged 54, who was under my care in this hospital for several weeks in the autumn of 1863, whose illness commenced three months prior to death with an injury to the abdomen, and in whose peritoneum there was also a large accumulation of slightly turbid and flaky fluid, with nodules of medullary cancer scattered over the peritoneal surface of the different viscera.

The characters by which you will recognize these cases are the following:

1. There are the phenomena of the cancerous cachexia, and perhaps a family history of cancer.
2. There is an accumulation of a large quantity of

fluid in the peritoneum, independently of disease of the liver, heart, or kidneys.

3. There is more or less fever, with great emaciation, vomiting, and severe pain and tenderness of the abdomen, approaching those of acute peritonitis. From acute peritonitis, however, the affection differs in the extensive effusion of fluid : in ordinary acute peritonitis also the quantity of fluid thrown out is small, but the tendency to collapse is greater, and the course more rapid.

It is not often that the nodules of cancer are large enough to be felt through the abdominal parietes.

IV. *Colloid Disease* occasionally causes great enlargement of the abdomen without any fluid in the peritoneum. You will find two cases of this sort recorded in the 'Pathological Transactions,' by Dr. Vanderbyl and Dr. O'Connor.* I show you here specimens of the colloid disease from the former case, which proved fatal in this hospital. But in many, probably the majority of cases, colloid disease, like ordinary cancer, leads to a large accumulation of fluid in the peritoneum either from exciting peritonitis or from the rupture of some of the colloid cysts and discharge of their contents, and paracentesis has been often resorted to, to relieve the breathing. The symptoms in these cases are very similar to those of primary cancer of the peritoneum. There is pain with tenderness in the abdomen, often preceding the signs of fluid for several weeks, and always accompanying them ; in most cases there is a certain degree

* Vol. ix. p. 207, and vol. xiii. p. 90.

of pyrexia and vomiting; but the swelling is much greater than in ordinary acute peritonitis. From the first there is rapid emaciation, and the case usually terminates fatally within six months of the commencement of the symptoms. As in cancer also, the disease is chiefly met with in middle and advanced life; of nine cases reported in the 'Pathological Transactions,' the ages varied from 37 to 75. But from cancer of the peritoneum colloid differs in these respects:

1. Even when the abdomen is very large, fluctuation is often very indistinct.

2. The fluid obtained by tapping is gelatinous, and sometimes so viscid that it will not flow through the cannula.*

3. Hard irregular masses or distinct tumours can frequently be felt through the abdominal wall.

4. Occasionally the patient voids per anum a gelatinous or slimy fluid, like white of egg.†

V. *Simple Dropsy of the Peritoneum*.—The causes of fluid in the peritoneal cavity already referred to being excluded, its presence must be due to simple dropsical effusion. This has the following characters:

1. There are the signs of fluid in the abdomen already enumerated (see p. 432).

2. There is an absence of fever or of persistent pain and tenderness of the abdomen.

* This happened in a case reported by Dr. Dickinson in the 'Pathological Transactions,' vol. xii. p. 92.

† Two cases are recorded in the 'Pathological Transactions' where this occurred—one by Dr. Quain (vol. iii. p. 319), and the other by Dr. O'Connor (vol. xiii. p. 90).

Simple dropsy of the peritoneum may have a three-fold origin. It may be due to :

1. Diseases of the kidneys.
2. Intra-thoracic diseases.
3. Diseases of the liver or portal vein.

Sir Thomas Watson mentions a form of dropsy which he calls *active ascites*, where fluid is rapidly thrown out into the peritoneum, after exposure to cold and wet, without fever or any sign of inflammation, and independently of any disease of the heart, liver, or kidneys, and where after a short time the fluid is again absorbed. Such cases must be very rare. The only case at all answering to this description which has come under my notice is that of a man who has been recently in this hospital, and in whom temporary ascites, without any fever, supervened upon an attack of what seemed to be colic (Case LXXXV.).

I. DROPSY OF THE PERITONEUM FROM DISEASES OF THE KIDNEYS.

The diseases of the kidneys which are most likely to induce dropsy are : *a*, acute nephritis, occurring as a sequel of scarlatina or from exposure to cold and wet, when the kidneys are hypertrophied and hyperæmic; *b*, the so-called 'large white kidney,' when the cortex is pale and hypertrophied, and the uriniferous tubes crammed with granular epithelium, and which is often merely an advanced stage of acute nephritis, but may also be developed independently as the result of a chronic inflammatory process; *c*,

the fatty kidney, where the organ is also large and pale, but the secreting cells are loaded with oil. Both clinical experience and post-mortem examinations make it probable that the fatty kidney is usually preceded by the 'large white' form of the disease. Either of these three diseases of the kidneys may give rise to ascites, which has the following distinguishing characters :

1. There is anasarca of the subcutaneous areolar tissue, which is *general* from the first, and is often noticed first in the face.

2. There is almost always evidence of fluid in the other serous cavities—the pleuræ and pericardium, as well as in the peritoneum.

3. The urine is scanty, turbid or smoky, and contains a large quantity of albumen, and often blood. Under the microscope you will usually find renal epithelium and casts of the uriniferous tubes varying in their character according to the particular disease in the kidney—epithelial, blood, and hyaline casts in acute nephritis; granular epithelial casts in the large white kidney; and oil-casts in the fatty kidney.

4. There is a pallor and pastiness of the countenance, which is almost pathognomonic.

5. There is a tendency to uræmia and the typhoid state, indicated by a dry brown tongue, fetid breath, loss of memory and restlessness, delirium, coma and convulsions.

All of these clinical characters you had an opportunity of studying in the case of James S——, who

was lately under my care in Cambridge Ward (Case LXXXVI.).

In forming a diagnosis it is necessary to remember that most renal diseases predispose to inflammation of the serous membranes, and that thus the symptoms of peritonitis may concur with those of renal dropsy, and also that the urine may contain albumen although the ascites is independent of disease of the kidneys (see pp. 247, 456).

Two common forms of chronic kidney-disease are rarely attended by dropsy and still more rarely by ascites. In the contracted, granular, or gouty kidney there is usually little or no dropsy at any stage of the malady; while in the waxy, or amyloid, kidney, anasarca rarely shows itself until shortly before the fatal termination, and even then is seldom excessive (see p. 26). Both of these diseases of the kidney are marked by the secretion of an increased amount of urine, and by a comparative absence from it of uriferous casts; but in the contracted kidney the specific gravity is low out of all proportion to the extent of dilution, the albumen is scanty or even altogether absent, and there is a marked tendency to uræmia; while in the case of the waxy kidney the lowness of the specific gravity of the urine is not more than may be accounted for by its dilution, the amount of albumen in it is large, and there is little tendency to uræmic symptoms, but in most cases there are the signs of waxy enlargement of the liver and spleen (see *antea*, pp. 24, 28).

II. DROPSY OF THE PERITONEUM FROM DISEASES IN THE CHEST.

Ascites is often a consequence of obstruction to the general circulation from various diseases of the chest, and especially from morbid conditions of the valves of the heart. Diseases of the mitral valve, and tricuspid incompetence whether secondary to mitral disease or to chronic bronchitis and emphysema, are more likely to cause dropsy and ascites than lesions of the aortic valves. A tumour compressing the inferior vena cava, above its junction with the hepatic vein, may lead to a like result.

Fluid in the peritoneum due to diseases obstructing the circulation of blood through the heart or lungs has the following characters :

1. Before there is any ascites there is anasarca, commencing in the feet and gradually proceeding upwards ; and even when the belly becomes swollen the swelling of the legs is large out of all proportion to the ascites.

2. There is a history of dyspnœa before any swelling shows itself in the abdomen. In extensive ascites, whatever be its cause, there is usually more or less dyspnœa, from the pressure of the fluid interfering with the action of the diaphragm and abdominal muscles. The peculiarity of the dropsy from intra-thoracic disease is that the dyspnœa precedes the ascites, and is distressing out of all proportion to its extent.

3. Intra-thoracic diseases which cause dropsy give rise at the same time to more or less lividity of the lips, face, and extremities.

4. There are the physical signs and symptoms of valvular disease of the heart or of old disease of the lungs. Even when the primary seat of obstruction is in the heart, there will be evidence of congestion or œdema of the lungs, or of bronchitis, pulmonary apoplexy, etc.

5. In the case of a tumour compressing the inferior vena cava there will be: *a*, the physical signs of the tumour; *b*, indications of its pressure upon other parts, such as the œsophagus or lung; *c*, a more rapid development and increase of anasarca than in ordinary cases of cardiac obstruction; and, *d*, great enlargement and tortuosity of the superficial veins of the chest and abdomen.

III. DROPSY OF THE PERITONEUM FROM DISEASES OF THE LIVER OR OF THE PORTAL VEIN.

The ascites which results from obstruction to the circulation through the trunk of the portal vein, or through its ramifications in the interior of the liver, is that with which at present we are more immediately concerned, and we shall therefore consider not merely the characters of the ascites from portal obstruction in general, but also those which pertain to the several causes of this obstruction. And in the first place, with regard to the distinguishing characters of the ascites from any cause of portal obstruction. They are as follows :

1. The dropsy in uncomplicated portal obstruction commences in the abdomen. The legs are only affected secondarily, and in consequence of the pressure of the ascitic fluid on the inferior vena cava. There are, however, exceptions to this rule. Patients will tell you occasionally that they have noticed slight swelling in the legs as soon as the abdomen began to enlarge. But in these cases there is this to be noted, that the swelling of the legs is slight out of all proportion to that of the abdomen, and that when the patient takes to bed it diminishes, or entirely disappears, while the ascites remains stationary or increases. Outgrowths from the liver, however, which compress both the portal vein and the inferior vena, may cause permanent dropsy both of the legs and abdomen. And lastly, there are these cases of chronic atrophy of the liver with portal obstruction which are secondary to diseases in the chest obstructing the general circulation, to which I have formerly adverted (p. 251). In such cases the ascites will be preceded by dropsy of the legs, and the addition of portal to general obstruction is not likely to be suspected unless there be a great preponderance of the dropsy in the peritoneum.

2. There is no sign of dropsy in the face, arms, or upper part of the trunk.

3. Dyspnœa often accompanies the swelling when this is great, but does not precede it, except in the one condition already referred to (p. 251).

4. Albuminuria is absent if there be no concurrent

kidney-disease. In reference, however, to this point, there is a common source of fallacy. The ascites itself, when extensive, may in consequence of the pressure of the fluid on the renal veins lead to the appearance of albumen in the urine, the albuminuria ceasing on the withdrawal of the pressure in the operation of paracentesis. At the same time you must remember that albuminuria will not admit of this explanation, unless the ascites be so great as to cause considerable tension of the abdominal parietes. The concurrence of puffiness of the face, or pitting of the arms or upper part of the trunk, or the presence of granular or oil-casts in the urine would also leave little doubt as to the existence of independent renal disease.

5. The ascites is accompanied by other indications of portal obstruction, such as enlargement of the spleen, enlargement and tortuosity of the superficial veins of the abdomen, hæmorrhoids, gastro-enteritis, and hæmorrhages from the stomach and bowels, which I have already described to you in a former lecture (see p. 244). The enlargement of the superficial veins of the abdomen is due to the collateral circulation established through the hæmorrhoidal plexus between the branches of the portal vein and those of the vena cava inferior, but is far from being a certain indication of the existence of portal obstruction. The same appearance is constantly observed in the ascites from disease of the heart, and sometimes as the result of pressure of a tumour or even of a large

quantity of ascitic fluid on the inferior vena cava; but in these cases there will be usually also a varicose state of the veins of the legs, although in the case of compression of the inferior cava, this may be in great measure prevented by great enlargement of the vena azygos. It must not be forgotten also that the epigastric veins, without being at all enlarged, may be rendered more visible in great ascites, owing to the stretching of the abdominal walls and the absorption of the subcutaneous fat.

6. Other indications of the existence of hepatic disease, such as enlargement, contraction, nodulation, or tenderness of the liver, jaundice, flatulence, &c., will often assist the diagnosis, but it is necessary to remember that all these signs may be absent or indefinite.

7. The fluid obtained by paracentesis is a clear, straw-coloured serum, containing a large quantity of albumen, but no urea, blood, or inflammatory products.

In the next place we have to inquire what are the diseases of the liver which are most likely to occasion ascites. And first it must be remarked that there are some morbid conditions of the liver which very seldom give rise to it. The fatty liver (see p. 45) and simple hypertrophy of the liver (p. 52), when uncomplicated, never cause it, and it rarely results from mere congestion (p. 119) unless this depend on mechanical obstruction of the circulation in the chest. Abscess (pp. 149, 169) or hydatid tumour (pp. 56, 110) of the liver do not lead to the presence of fluid in the peri-

toneum, except in rare cases, from the direct pressure of the tumour on the trunk of the portal vein, from the bursting of the cyst, or through the intervention of peritonitis.

Fluid in the peritoneum is a more common accompaniment of the waxy or amyloid liver (p. 26) than of any of the maladies just named, but even here it is rare. Frerichs noted it in only eight out of 23 cases, and in four of the eight cases it was due to the supervention of acute peritonitis.* When its origin is not inflammatory, it is probably due to enlarged waxy lymphatic glands in the fissure of the liver pressing upon the trunk of the portal vein (see Case LXXXIX.), or to the concurrence of peritonitis or cirrhosis (pp. 26, 42). The freedom from ascites in waxy liver is accounted for by the fact that it is the branches of the hepatic artery, and not of the portal vein, which are implicated in the disease. Fluid in the peritoneum from waxy liver will be recognized by the following characters (see also p. 24) :

1. Uniform (but in very rare instances, nodulated, pp. 26, 193), solid, painless enlargement of the liver.

2. Enlargement of the spleen.

3. A large quantity of albumen in the urine, with little or no dropsy, and those other characters of the urine which I described to you in my lecture on waxy enlargement of the liver (p. 28). Albuminuria, however, is not necessarily present in cases of waxy disease. Since my lecture on the subject was delivered,

* Diseases of the Liver. Syd. Soc. Ed. vol. ii. p. 179.

you have seen two cases in the wards, where there was considerable waxy enlargement of both liver and spleen, but where the kidneys were so slightly implicated that the urine contained no albumen.

4. Great anæmia.

5. A history of disease of the bones, or joints, suppurating sores, phthisis, constitutional syphilis, &c.

The diseases of the liver which most commonly give rise to portal obstruction with ascites are :

1. Cirrhosis and the other forms of chronic atrophy of the liver.

2. Cancer of the liver.

3. Peri-hepatitis.

4. Thrombosis or obstruction of the trunk of the portal vein.

Practically, it is chiefly with the two first that you will have to deal, as the others are of much rarer occurrence.

1. *Ascites from Chronic Atrophy of the Liver.*

Cirrhosis and the other forms of chronic atrophy of the liver are far the most common causes of portal obstruction leading to ascites. In all these cases the liver is contracted and the area of hepatic dulness is diminished. The clinical characters of ascites from chronic atrophy of the liver are as follows :

[See *antea*, pages 243 to 253.]

2. *Ascites from Cancer of the Liver.*

Cancer of the liver is often attended by fluid in the peritoneum, but the quantity of fluid is usually small

as compared with what is observed in cirrhosis ; while the liver, instead of being contracted, is enlarged. In many cases the ascites has an inflammatory origin, and is attended by pain, tenderness, and other symptoms of peritonitis during life, while the fluid in the peritoneum contains flakes of lymph, or even blood, owing to rupture of the capsule covering some of the deposits of cancer.

The liver may be greatly enlarged from the presence of isolated nodules of cancer without any ascites, the portal circulation being sufficiently maintained by the healthy glandular tissue intervening between the cancerous nodules. Ascites under these circumstances is only produced when the trunk of the portal vein is compressed in the portal fissure by cancerous lymphatic glands, by a cancerous outgrowth from the liver itself, or by new connective tissue resulting from perihepatitis (see Case XLII., p. 198). But in the infiltrated form of cancer the ramifications of the portal vein in the interior of the liver are destroyed as the disease advances, and hence, when this is at all extensive, there is almost always ascites.

The clinical characters of ascites from cancer of the liver are as follows :—

[See *antea*, pages 187 to 193.]

3. *Ascites from Peri-hepatitis.*

When lymph thrown out into the portal fissure becomes organized, it may cause constriction of the trunk of the portal vein, and all the phenomena of

portal obstruction. As a rule, this development of new connective tissue extends over the entire surface of the liver and also into its interior, and there is developed that condition of the organ known as ‘ Simple induration,’ which has been already described to you as one of the forms of chronic atrophy (pp. 241, 252). I have nothing now to add as to the clinical characters of peri-hepatitis, except that it may occasionally cause ascites without any contraction of the liver, and that when the peri-hepatitis has been excited by cancer in the liver, the organ may be even enlarged.

4. *Ascites from Thrombosis or Obstruction of the Trunk of the Portal Vein.*

Coagula are liable to form in the portal vein and obstruct its passage from various causes.

a. Disease of the coats of the vein may lead to coagula in its interior. It was at one time contended that coagula here, as in other parts of the venous system, were almost always the result of inflammation of the venous coats, or phlebitis, but with our present knowledge it is doubtful if the inflammation of the venous wall (indicated by thickening, adhesion to its contents, etc.) be not often the consequence rather than the cause of the coagulation. There are, however, a considerable number of cases on record where calcification of the wall of the portal vein has converted the vein into a rigid narrow tube, which has then become suddenly blocked up by coagulum. You will find an interesting case of this sort reported by Dr.

Andrew Clark, in the last volume of the 'Pathological Transactions' (vol. xviii. p. 61), and references to many others have been collected by Frerichs.*

b. Many cases of thrombosis of the trunk of the portal vein are secondary to diseases of the glandular tissue of the liver, which obstruct or destroy the branches of the vessel, such as the various forms of chronic atrophy and infiltrated cancer. The obstruction in these cases commences in the ramifications, and extends to the trunk, of the vein.

c. Thrombosis of the portal vein may be induced by compression of the vein from without by enlarged cancerous (pp. 191, 460) or waxy (pp. 26, 458) glands, connective tissue from old peri-hepatitis (pp. 241, 460), outgrowths from the liver, tumours of the pancreas, omentum, etc. If the compressing force be great, the vessel may be flattened and its walls brought into apposition; more commonly the compression acts by retarding the circulation and favouring coagulation.

d. Lastly, there are some cases of thrombosis of the portal vein which seem to result from mere weakness of the circulation, or from an unusual disposition of the blood to coagulate.

The symptoms of thrombosis of the trunk of the portal vein are those already described to you (p. 244) as resulting from obstruction or obliteration of the ramifications of the vessel in chronic atrophy, but in an exaggerated form. They are :

1. The rapid development of extreme ascites, often

* Op. cit. vol. ii. p. 402.

necessitating paracentesis to avert asphyxia, and returning immediately after the operation.

2. Rapid enlargement of the veins of the abdominal wall, which resemble large cords.

3. Urgent vomiting and diarrhœa.

4. Copious hæmorrhage, in many cases, from the stomach and bowels. Occasionally this is the first and chief symptom, and the patient dies from syncope, before there is time for ascites, &c.

5. Great enlargement of the spleen.

6. Obstruction of the trunk of the portal vein in most cases is speedily fatal, but if it be not, it leads to atrophy of the liver.* A contracted liver may thus be either the cause or the consequence of portal obstruction.

I have thus endeavoured to lay before you succinctly, and perhaps somewhat dogmatically, the distinguishing characters of fluid in the peritoneum according to its several causes. In practice, however, you must remember that there may be a concurrence of different causes in the same case. For instance, I have more than once pointed out to you that portal obstruction may be secondary to obstruction of the circulation in the heart or lungs, and I have also shown you that ascites may result from either of these causes independently. Secondly, although, as we have seen, albuminuria may be induced by the

* See, for instance, a case reported by Dr. Dickinson in the 'Pathological Transactions,' vol. xiv. p. 63.

ascites itself (p. 456), it is quite possible, and indeed not uncommon, to have disease of the kidneys concurrent with portal obstruction. Spirit-drinking, which is so fruitful a source of cirrhosis of the liver, contributes also to the development in many cases of nephritis and of the fatty kidney. Lastly, thrombosis of the trunk of the portal vein is often a result of obstruction of the ramifications of the vessel in the interior of the liver, and when this occurs, the symptoms of slight portal obstruction may be suddenly succeeded by those of complete obstruction.

The Treatment of Fluid in the Peritoneum.

The proper treatment for any case where there is fluid in the peritoneum must depend entirely on the cause of this condition, and you will therefore perceive the necessity which exists of being able to recognize the cause, or the predominant cause, in each particular case. It would be out of place on the present occasion, when I wish to direct your attention mainly to the diseases of the liver, to enter into a consideration of the proper treatment for the various maladies that may give rise to fluid in the peritoneum; and I have already described to you in some detail the appropriate treatment for the ascites and other symptoms resulting from portal obstruction. It is unnecessary to recapitulate the remarks on this matter, made in my lecture on Chronic Atrophy of the Liver (p. 255).

It may, however, serve to impress more durably on your minds the remarks which have now been made

on the distinguishing characters of fluid in the peritoneum according to its several causes, and also the rules laid down in my systematic lectures on Medicine, for the treatment of the various maladies from which ascites may arise, if I bring under your notice the particulars of a few cases which, with two exceptions, you have had an opportunity of watching in the wards.

Case LXXIX. illustrated at first the points of distinction of fluid in an ovarian cyst from ascites or fluid in the peritoneum; but subsequently the physical signs were remarkably modified by the entrance of air into the ovarian cyst. A mistake in diagnosis was all the more likely to have been committed by a careless observer from the concurrence of albuminuria and general dropsy. The case is remarkable from its rare mode of termination, the ovarian cyst opening into the rectum. No similar case is reported in the entire series of the 'Pathological Transactions:' in the fourteenth volume (p. 201) Dr. Bristowe has recorded a case where there was a communication between the rectum and an ovarian cyst, but in that case there was extensive tubercular ulceration of the bowel, and the perforation advanced from the bowel to the ovary. In the case of Elizabeth C——, you will remember that we were enabled to diagnose not merely the existence of the ovarian tumour, but also the fact of the cyst having burst into some portion of the bowel. After removal from the body, the walls of

the cyst collapsed and shrivelled, so that no adequate idea of its size prior to bursting can be gathered from the preparation which I now show you.

CASE LXXIX.—*Cystic Tumour of the Ovary opening into the Rectum—Entrance of Air into the Ovarian Cyst—Atrophy of Right Lobe of Liver and complementary Hypertrophy of Left Lobe.*

Elizabeth C—, aged 37, was admitted into the Middlesex Hospital under my care on August 23, 1866. She had been married twice, but only had one child still-born (1852), and never any miscarriages. The catamenia were regular, the last period having ceased the day before admission. At the age of 16 she had been laid up for six weeks with scarlet fever, but she did not know if she had dropsy. Ever since she had suffered from pains in her back, and for the last eight years she had been liable to general dropsy and attacks of erysipelas of the face. Eighteen months before admission, she had first noticed a swelling in the lower part of the abdomen, which had been slowly increasing.

On admission, the abdomen was found considerably distended by a tumour rising above the pubes and reaching to above the umbilicus. This appeared to occupy a middle position in the abdomen, but could be traced more readily into the left inguinal region than into the right. The tumour was dull on percussion and distinctly fluctuating; behind it, in either flank, percussion yielded a tympanitic sound. Both lower extremities were much swollen, œdematous, and tender, and the face was slightly puffy. The cardiac and respiratory signs were normal, but the areæ of hepatic and splenic dulness were increased. The urine contained a considerable amount of albumen, and deposited epithelial and oily casts; its spec. gr. was 1016. The pulse was 96 and feeble, and there was occasional vomiting. On August 25 the patient began to suffer from diarrhœa; the motions contained blood, and there was considerable tenderness of the abdominal tumour.

On August 31 the diarrhœa continued; there was no pain in defæcation; the tongue was clean and too red, and the breath was very offensive.

On September 10 there was no abatement of the diarrhœa,

notwithstanding the free exhibition of astringents. The tongue was dry and brown and the breath extremely offensive. The patient was entirely prostrate, drowsy (from opium?), and occasionally delirious. There had been no rigors or sweating, and there was no diminution in the size of the tumour, the girth of the abdomen being the same as at the time of admission (thirty-six inches).

On September 11 the motions were observed to contain a quantity of pus, which continued to be passed for three days, and on September 17 all signs of the tumour had disappeared, the percussion sound above the pubes being equally tympanitic as in the flanks.

After this there was little diarrhoea, but the patient continued to sink, and died on September 19.

At the *autopsy* a thick layer of fat was found beneath the skin (half an inch over the abdomen). The heart and lungs were healthy. The liver weighed fifty-three ounces; and the right lobe was much atrophied and deeply lobulated; but the left lobe was enormously increased, being nearly three times the size of the right; its structure appeared healthy. The spleen weighed twenty ounces, was very firm, and presented the typical characters and reaction of waxy degeneration. The two kidneys weighed together eighteen ounces and a half; both were pale and smooth, pale-yellow and opaque; their cortex was greatly hypertrophied and their secreting cells loaded with oil.

On first opening the abdomen, no tumour was visible. The intestines came down to the pubes; but on raising a few coils, a collapsed cyst about the size of a cocoa-nut was seen in the situation of the uterus. On further examination, this was ascertained to be a cyst of the left ovary, which had emptied itself by an opening the size of a fourpenny piece into the rectum four inches above the anus. Its walls were fibrous and about half an inch thick, and it contained a little dirty, very fetid pus. The sigmoid flexure of the colon took a turn transversely to the right side across the upper part of the tumour, to which it was firmly adherent. The free end of the appendix vermiformis also adhered to it. There was no ulceration of the mucous membrane of the rectum round the opening into the ovarian cyst.

The following very remarkable case illustrates the

difficulty in diagnosis which may arise from great enlargement of the urinary bladder. In a female the physical signs would have at once suggested an ovarian tumour, but in a male they seemed only explicable on the supposition of an hydatid of the liver, and it was with this view that the surgeon under whose care the patient was had recourse to paracentesis. The great size of the swelling, the patient's statement that it commenced above the umbilicus, the fact of the greatest girth of the abdomen being three inches above the umbilicus, and the complete freedom from urinary symptoms, suppressed all suspicion of the bladder being primarily at fault. Such a case is not likely ever to occur in a female, and the lesson which it teaches is that whenever you find the physical signs which were present in this case, and especially when they occur in an aged male, the first thing to be done is to introduce a large prostatic catheter into the bladder and to push it well up. It is worth observing, however, that in this case the puncture was not followed by any extravasation of urine or sign of peritonitis, and that the immediate cause of death appeared to be passive hæmorrhage from the mucous membrane of the bladder in consequence of the withdrawal of the urine.

CASE LXXX.—*Enlargement of the Abdomen from a distended Urinary Bladder mistaken for an Hydatid Tumour of the Liver—480 ounces of Urine drawn off by Paracentesis Abdominis.*

Mr. F——, a feeble-looking elderly gentleman, of small frame and spare habit, consulted an eminent surgeon, to whom I am indebted for these particulars, at the beginning of June 1866. He stated that until three years before he had always enjoyed perfect health and been engaged in active pursuits. He then first noticed a swelling *above the navel*, which continued to increase, but caused him little inconvenience until the beginning of the previous March, when his breath became short and he began to lose flesh and strength and to get nervous. Early in May his left thigh and leg became swollen and pitted on pressure, but after two weeks the swelling disappeared. For several weeks he had been troubled with frequent vomiting after food. He had nevertheless been able to ride for several hours a day up to the time of his presenting himself for advice. His condition was then noted to be as follows:— ‘He presents the appearance characteristic of abdominal disease. Face thin and cadaverous, but not jaundiced. The abdomen is enormously enlarged, being occupied by a tumour of uniform surface and oval form, of which the narrow end is uppermost. The ensiform cartilage and the lower ribs on both sides are elevated, and the tumour appears to spring from beneath them and to fill up the entire anterior portion of the abdomen. Fluctuation is distinct throughout every part of it, but it is particularly marked above the umbilicus. It is everywhere dull on percussion, but in both flanks, in whatever posture the patient assumes, there is tympanitic percussion-sound. The abdomen is largest three inches *above* the umbilicus where its girth is 43 inches; the distance from the pubes to the ensiform cartilage measures 21 inches. A few blue veins are seen coursing over the abdominal wall. Heart sounds weak, but in other respects normal. Urine abundant; sp. gr. 1010; faintly acid, and with a slight trace of albumen. He has passed water three or four times a day, and once during the night, always in a full stream and with perfect relief.’ The patient always insisted that he had never suffered the slightest urinary symptoms or trouble in micturating, but subsequently, after the bladder had been tapped, he admitted that for some years he had made a practice of sitting down when he made water, because he had found that the urine ‘came better’ when he did so.

On June 5 the abdomen was tapped with a fine trocar midway between the umbilicus and the ensiform cartilage. Twelve quarts or 480 ounces of fluid drained away, the time occupied being two hours. The fluid was of a pale straw colour; sp. gr. 1010, feebly acid,

slightly albuminous, and had a urinous odour. A portion sent to me for examination contained urea and a few blood corpuscles.

June 6. Patient has had a comfortable night, but has passed no urine since the operation. Half a pint of *dark* urine was drawn off by catheter in the morning, and $1\frac{1}{2}$ pint in the evening. The abdomen is again large.

June 7. Has had another good night and has no symptom of constitutional disturbance. A pint and a half of dark urine was drawn off in the morning, and at 3 P.M. *six pints* more, and as this flowed away the swelling again disappeared, and the patient's belly became quite flat. The catheter was tied in, but soon slipped out. During the hour occupied in emptying the bladder, the patient felt very faint and soon afterwards he had a rigor and fainted on getting up to try to micturate. Stimulants were given freely and the fainting did not return, but he had a restless night and vomited several times.

June 8. The urine was drawn off twice—morning and evening—and was observed to contain much blood. During the day he vomited four times. He was ordered gallic acid and ice.

June 9. Patient much weaker and the urine drawn off by catheter is almost black from the amount of contained blood. About 8 P.M. he passed into a state of collapse, which continued until death, at 3 A.M. on June 10.

Autopsy.—There was no evidence of peritonitis. The bladder lay collapsed and flaccid in front of the intestines, filling up nearly all the front part of the abdomen, and its apex being within $1\frac{1}{2}$ inch of the ensiform cartilage. The reflection of peritoneum from the bladder to the abdominal wall was within one inch of the umbilicus. A small red spot, like a flea-bite, near the fundus, corresponded to the puncture. The bladder contained a considerable quantity of bloody urine. The muscular coat was thickened; the mucous membrane was thrown into prominent folds, which were very red and congested, the spaces between being pale. The ureters were dilated and the kidneys sacculated: in the left kidney very little secreting tissue remained. The prostate was enormously enlarged, almost filling the pelvic cavity.

In the next case the peritoneum contained fluid as the result of acute peritonitis.

CASE LXXXI.—*Fluid in Peritoneum from Acute Peritonitis due to a Kick over a Congenital Hernia.*

Herbert R—, aged 12, was admitted into the Middlesex Hospital under my care on October 28, 1866. Since four years of age he had been known to have an inguinal hernia on the right side, but this had never caused him much inconvenience; and, excepting the infectious diseases of childhood, he had enjoyed good health until two days before admission. On the morning of October 26 he received a kick while in bed over the right testicle. This was followed by considerable pain in the testicle, but he got up and went to school. In the afternoon he had a rigor lasting for half an hour, followed by diarrhœa, and later in the evening by vomiting and pain at the epigastrium, extending thence over the whole abdomen. During the whole of the 27th and the following night he had frequent rigors and vomiting, and urgent diarrhœa with light-coloured stools.

On admission, pulse 144; temperature 104°; respirations 34 and thoracic; abdomen distended and extremely tender, especially in the right groin; the right testicle was much enlarged and exquisitely tender; cardiac and pulmonary signs normal. The boy was deaf and confused in his mind, like a patient suffering from fever. Poultices with laudanum were applied to the abdomen, and within the first 24 hours after admission the patient took as much as five grains of opium internally. Under this treatment the acute symptoms subsided—the pain and vomiting were diminished and the diarrhœa ceased; but no material improvement took place. The tongue became dry and brown; the prostration increased; the cheeks were sunken and the features pinched; and there was occasional delirium.

On October 30 the temperature was normal (98°), but the pulse was 132; the abdomen was more distended, and there was distinct evidence on tapping of fluid in the peritoneum. The bowels were confined, but the vomiting had returned. On November 1 the temperature was still 98°, but there was no improvement in the patient's general condition.

The diagnosis in this case, as frequently stated at the bed-side, was that the patient was suffering from acute peritonitis excited by the kick on the scrotum, the inflammation being propagated

to the peritoneum, from the hernial sac. It was suggested, however, by a gentleman who accompanied me on my visits, that possibly a portion of bowel had become strangulated in the neck of the hernial sac; but although this view was favoured by the fact that the bowels had not acted since the boy's admission, the bowels had been very relaxed previously and the constipation was accounted for by the opium that had been taken subsequently. One of my surgical colleagues, however, who at my request saw the patient in my absence, thought that probably there was a portion of bowel or omentum strangulated in the sac, and cut down upon it. A little pus escaped, but none of the intestinal contents were found in the sac. The boy gradually sank, and died at 8 A.M. on the following morning.

Autopsy.—The chief morbid appearance was very extensive recent peritonitis, the surface of the liver and the whole of the bowels being plastered over with soft yellow lymph. The peritoneum also contained two or three pints of purulent fluid. There was no ulceration, perforation, or gangrene of any portion of the stomach, bowel, or appendix vermiformis, to account for the peritonitis. There was no bowel or omentum in the hernial sac, or even adherent in the neighbourhood of the internal opening. There was intense vascular injection of the outer surface of the right testicle.

In the following case fluid was thrown out into the abdomen as the result of sub-acute peritonitis. But the chief pathological interest of the case lay in the fact that the appearances found in the liver after death corresponded in every way with those which have been so often described of late years as constituting one of the lesions of constitutional syphilis (see p. 241), and yet that the evidence was as strong as negative evidence can well be in such a matter, against the view that the patient had ever suffered from syphilis.

CASE LXXXII.—*Fluid in Peritoneum from Chronic Peritonitis—Chronic Atrophy of Liver with Fibroid Nodules in its interior, apparently independent of Syphilis.*

P. D——, aged 67, was admitted into the Middlesex Hospital under my care on January 17, 1867. He was a labourer, had been married for 23 years, and was the father of seven children. His oldest child was 22; all were in good health, and none had died; his wife had never had any miscarriages. A brother had died of consumption, but the patient himself had always enjoyed good health, except for about three months three or four years previously, during which time he had been in hospital for a fracture of the arm and other injuries. He never had suffered from syphilis, rheumatic fever, dropsy, jaundice, vomiting, or hæmorrhoids. His habits had been temperate. Six weeks before admission, a heavy weight fell upon his head, which wounded the scalp, and stunned him for a few seconds. For four days after this he gave up his work, and complained of pain in the region of the liver. On the fifth day, he returned to his work, but after a few hours, he was obliged to give it up. A week after the accident, he noticed that the abdomen was swelling, and a fortnight after, he began to vomit his food as soon as it was swallowed. Between this date and that of admission he had become very emaciated.

At the time of admission, the patient was greatly emaciated, and his countenance was expressive of suffering. The tongue was moist and coated with a yellowish fur. He had no appetite, but complained of thirst, although he was afraid to drink, as everything he took was rejected within a quarter of an hour. He distinctly stated, however, that he had no pain between swallowing and vomiting. The abdomen was considerably distended, the girth at the umbilicus measuring $33\frac{1}{2}$ inches, and there was unmistakable evidence of fluid in the peritoneum. The hepatic dulness in the right mammary line measured only three inches; there was no jaundice. The splenic dulness was not increased, and there was no enlargement of the subcutaneous veins of the abdomen. Immediately above and below the umbilicus there was an obscure induration, with no defined margin, and, at some parts, yielding clear percussion. The abdomen generally was slightly tender, but by no means acutely so, and there was fair movement of the abdominal muscles

in respiration. The patient stated that he was not in much pain, but he seemed to be always easier when lying on his back with his legs drawn up. The bowels were regularly open. The pulse was 96 and regular; the cardiac dulness was less than natural, and the sounds were normal. The respirations were 26, and there was coarse crepitus over the bases of both lungs, without any dulness or tubular breathing. There was not the slightest œdema of the legs, trunk, or face, and the urine was free from albumen.

All efforts to relieve the vomiting proved unavailing. The size of the abdomen remained stationary. On January 20, hiccup came on, and, notwithstanding the frequent exhibition of nutritious enemata, the emaciation rapidly increased until January 28, when the patient died from exhaustion.

At the *autopsy*, one gallon of turbid fluid was found in the abdominal cavity in front of the small intestines, which were firmly matted together, forming a globular mass pointing towards the umbilicus, and accounting for the obscure tumour felt during life. The peritoneum was everywhere coated with a thick layer of reticulated lymph, and the great omentum was much thickened and indurated, but nowhere about the bowels or mesenteric glands was there any indication of tubercular or cancerous deposit. The stomach was much contracted, and its mucous membrane for several inches from the pylorus was reddened and thrown into folds, and under the microscope presented a remarkably villous appearance. The pyloric end of the stomach was surrounded and pressed on by greatly thickened omentum; but nowhere in its coats could any appearance or structure resembling that of cancer be discovered. The liver was small, weighing only 40 ounces. Its capsule was at some places thickened and adherent to surrounding parts, and over its surface were several cicatrix-like depressions. Scattered through the substance of the liver were numerous rounded opaque-yellow deposits, the largest about the size of a cherry. Such of the deposits as were immediately beneath the capsule were not at all raised above the general surface, while others were situated at the bottom of the cicatrix-like depressions. On section they presented a firm, fibrous-looking appearance, and yielded no milky juice. On microscopic examination they were found to be made up of white fibrous tissue, with nuclei and small fibre-cells and granular matter, but to contain nothing suggestive of cancer. The spleen was small; the lungs were congested and

oedematous at their bases; and the kidneys were slightly granular. The heart was small, but healthy. No deposits like those in the liver could be found in any other organ, and no cicatrices could be discovered on the penis, in the groins, or on the legs.

Case LXXXIII. is a good example of fluid in the peritoneum from cancerous peritonitis.

CASE LXXXIII.—*Primary Cancer of the Peritoneum, causing a large effusion of Fluid.*

Jane A——, aged 51, a nurse in a private family, was admitted into King's College Hospital under my care on July 22, 1859. Her general health had always been good prior to the illness for which she was admitted. She had never had any other illness of importance, and had always lived comfortably and been temperate in her habits. No hereditary tendency to cancer could be ascertained.

Three months before admission she began to complain of pain in the lower part of the belly, principally in the situation of the left ovary and of the bladder, and also in the back. The pain in the bladder was always worse after micturition. She was thought to have inflammation of the bladder; leeches were applied and the pain abated. Still she kept at her work until a fortnight before admission, when she was seized somewhat suddenly with febrile symptoms, vomiting, much pain and tenderness of the abdomen, followed by swelling, which rapidly increased.

On admission the patient was emaciated, but the abdomen was greatly distended, measuring $36\frac{1}{2}$ inches at the umbilicus, and presenting all the characters of fluid in the peritoneum with the addition of great tenderness on pressure, especially on the left side. The pulse was 92. The tongue was very red and clean; the bowels constipated, but easily acted on by medicine; and occasionally there was much vomiting. No enlargement could be discovered either of the liver or spleen, and there was no jaundice. There was no anasarca of the legs, and the heart's sounds were normal. The urine was scanty and dark, but contained no albumen. The treatment consisted in the use of effervescing draughts with hydrocyanic acid to allay the sickness, a diuretic pill containing squill,

digitalis and blue pill, and colocynth and henbane pills to keep the bowels open.

At first the diuretics increased the flow of urine, and the size of the abdomen remained stationary; but about the middle of August they seemed to lose their effect, the abdomen became larger, while the tympanitic portion, which always was uppermost in whatever position the patient lay, became smaller. On August 27 the abdomen measured 40 inches in circumference, and the patient complained much of its feeling very tight and painful. The respirations were 38 and thoracic, and there was much dyspnoea from the pressure of the fluid on the diaphragm; there was nothing abnormal in the physical signs of the lungs. The tongue was still very red and clean; the vomiting was more frequent and urgent; the bowels were never opened without medicine. From the first she had continued losing flesh, and her features were now pinched, and she slept little. To-day the abdomen was tapped, and about two gallons of a transparent greenish-yellow fluid, having a specific gravity of 1020, containing much albumen, and also many white flakes of fibrillated lymph, were drawn off.

For two days afterwards the patient experienced great relief; the sickness and pain ceased. No tumour could be felt in the abdomen after the evacuation of the fluid. On the morning of August 30, the patient was seized with severe vomiting, and a return of pain and tenderness in the abdomen, which was much distended and tympanitic, while at the same time there was evidence of a small quantity of fluid in the peritoneum. The vomiting, emaciation, and abdominal pain continued, uninfluenced by treatment. The patient could bear nothing in her stomach except champagne and ice, and for four weeks she was nourished by enemata of beef-tea, eggs, and brandy, with a few drops of laudanum. Aphthæ appeared on the tongue. The fluid did not reaccumulate in large quantity in the abdomen, but early in September several small nodules could be felt through the abdominal parietes. On September 18, the left thigh and leg were noted to be swollen, and there was tenderness along the course of the femoral vein. After this the patient was thought on several occasions to be moribund; but she rallied, to die at last on September 28, in a state of extreme emaciation.

Autopsy.—Only 30 ounces of clear straw-coloured fluid were found in the peritoneum. The peritoneal surface of all the intestines, of the liver, and of the bladder, were studded over with in-

numerable nodules of cancer varying in size from a pin's head to a hazel-nut. The mesenteric glands were also slightly enlarged from cancerous deposit. The cancerous masses exuded a milky juice on section, which contained characteristic 'cancer-cells.' The intestines were connected here and there by a few loose adhesions, and the sigmoid flexure was firmly bound down over the iliac vein by firmer adhesions and nodules of cancer. There was no cancer of the mucous or muscular coats of the stomach or bowels, or in the uterus, liver, kidneys, or lungs, but in the apex of the right lung were several cretaceous nodules. The liver and spleen were of normal size. The left iliac and femoral veins were plugged by adherent coagula.

In the following case there was a circumscribed collection of fluid in the peritoneum resulting from tubercular peritonitis. In this disease the abdomen is usually retracted and there are no signs of fluid, and the exceptional appearances in the case of Thomas M—— were no doubt due to perforation of the bowel and the formation of a circumscribed peritoneal abscess. You will remember that this was the view taken of his case while the patient was in the hospital under your observation.

CASE LXXXIV.—*Tubercular Peritonitis—Signs of a circumscribed collection of Fluid in Peritoneum.*

Thomas M——, aged 32, a labourer, came from Lincolnshire to be admitted into the Middlesex Hospital under my care on September 11, 1866. For four years he had been a 'teetotaller,' and he had never in his life drunk spirits except what had been ordered for him during his illness. With the exception of slight cough and hæmoptysis, two years before, he had been always well and hearty until six months before admission, when he began to suffer from slight diarrhoea,—about three motions a day. After six weeks he felt weak and was obliged to give up work, and about a month after this he noticed a swelling in the lower part

of the abdomen, which continued increasing until about six weeks before admission, when it became rather suddenly reduced and since then had been stationary. The reduction in size had been accompanied by a considerable increase in the purging. From the commencement of his illness he had lost flesh, and for six weeks his appetite had failed and he had suffered from oppression at the stomach after meals.

On admission, the patient was emaciated and anæmic. The pulse was 84; the cardiac and respiratory signs were normal. The urine contained no albumen, but threw down a copious deposit of lithates, and became dark red on adding nitric acid after boiling. The patient's disease seemed confined to the abdomen, in the lower part of which there was a uniform prominent bulging extending to an inch above the umbilicus, equal on the two sides, but yielding a clear percussion-sound on the right, and on the left complete dullness with a distinct thrill on gentle tapping, and sometimes gurgling on pressure. The relative positions of the dull and clear spaces were not altered by posture. There was slight tenderness on pressure over the swelling, especially at its circumference, which was distinctly defined and indurated. Above it the abdomen was quite flat. The hepatic dullness in the right mammary line was only three inches. The tongue was clean, and not abnormally red. He always complained of pain in the abdomen after drinking cold fluids. The bowels were open four or five times a day, the motions being yellowish-brown and thin.

The patient was treated first with acetate of lead and opium and chalk mixture and catechu, with milk diet and port wine, and subsequently he took tincture of iron with opium pills. The purging, however, was never completely checked; he had from two to twelve motions a day, and he was always worst when the opium was omitted. On October 1, he had rather a severe attack of pain in the abdomen followed by increased purging and a slight diminution in the size of the tumour. About the middle of October he began to complain of cough and nocturnal perspirations and decided dullness was noted below the right clavicle. Pulse 100. Temperature 100·2°. He was now ordered quinine, sulphuric acid, and opium.

About the beginning of November his feet and ankles became œdematous and about the same time a change commenced in the abdominal swelling, from which the signs of fluid disappeared, while

the whole swelling became smaller, so that the girth of the abdomen below the navel, which on admission had been 32 inches, was reduced to 29 inches on December 28. The diarrhœa, however, continued, and the patient got weaker, although he was still able to go about the ward. On January 2, the dulness below the right clavicle was ascertained to be much more extensive and decided, and there was also marked flattening there, with prolonged expiration and crepitation. He had also frequent retching after food. On February 28, he was discharged from the hospital at his own request, and went back to Lincolnshire. The swelling in the abdomen was now almost gone, but in its site there was considerable induration and tenderness.

On April 10 he died, after an attack of erysipelas of the left leg, and Dr. W. J. Hodgson, of Long Sutton, was good enough to send me an account of the post-mortem examination, with specimens of the morbid structures. There were extensive tubercular deposits and large vomicæ in the upper part of the right lung, and there was also extensive deposit of tubercle throughout the rest of the right, and the whole of the left, lung. The intestines were everywhere firmly matted together by tubercle and organized lymph, particularly in the left inguinal region, where the bowels ruptured on attempting to separate them, disclosing cavities which contained fæces and pus and communicated by perforations with the bowels, in the mucous membrane of which were numerous tubercular ulcers. The mesenteric glands were enlarged from tubercular deposit, and there was also much tubercular induration of the great omentum, which corresponded to the hard boundary of the tumour felt during life.

The next case is a very remarkable one. It is that of a patient who has recently been in the hospital and who had for a time unmistakable signs of fluid in the peritoneum, the cause of which was not very apparent. Notwithstanding the unusual degree of tenderness, the slow pulse, the low temperature, and the paroxysmal character of the pain led us to regard the case, in the first instance, as merely one of severe colic.

CASE LXXXV.—*Symptoms of Colic followed by signs of Fluid in the Peritoneum.*

Edward J——, aged 21, who had formerly been a printer, but had been working for six weeks at a carver and gilder's, was admitted into the Middlesex Hospital under my care on April 12, 1868. On April 6, he had been suddenly seized with severe pain in the abdomen and retching. The pain had been constant ever since, but had been also liable to severe exacerbations. The vomiting had recurred daily, but had not been so violent as at first. The bowels had acted on the 8th and 10th after castor oil and laudanum. Shortly before his attack the patient had been suffering from gonorrhœa, and he stated that some years before he had a similar, though much less severe, attack of abdominal pain.

On admission, the patient still complained of constant pain in the abdomen, with frequent acute exacerbations. The pain was increased by any movement, and there was also considerable tenderness over the abdomen, most marked over the cœcum. The abdomen was distended and tympanitic, and the breathing was entirely thoracic. There was frequent retching of scanty bilious matter. There was a dark red (not blue) line along the margin of the gums. The tongue was moist and only slightly furred; there was thirst, and the bowels had not been open for two days. The pulse was 84; the skin was cool; and the temperature under the tongue 97°. There was no albumen in the urine. The patient was ordered a warm bath, warm fomentations to the belly, an enema of three pints of barley-water with four drachms of tincture of assafoetida, and a grain of opium every four hours.

The enema brought away two copious motions, but with no relief to the pain. On April 13, a third of a grain of extract of belladonna was ordered every three hours, but next day the pain, tenderness, and tension of the abdomen had increased, although the pulse was only 72, and the temperature 97°. He was again ordered a grain of opium every four hours, a draught of castor oil and laudanum, and frequent enemata. He also continued taking six grains of opium a day until April 17, and then three grains until April 23. Under this treatment the bowels were freely moved, and the paroxysms of pain became less severe; but he still had occasional vomiting, the abdomen grew larger and more tense, and

on April 19 there was unmistakable evidence of fluid in the peritoneum. A thrill could be propagated from one side to the other on tapping, and when the patient was supine there was dullness in either flank, which varied with his posture. He still had occasional paroxysms of pain, but no tenderness of the abdomen. The pulse, however, kept steadily at 72, and the temperature rarely exceeded 98°. The signs of fluid in the peritoneum, with occasional slight paroxysms of pain, continued until May 4. After this the abdomen gradually became smaller, and on May 18 it had regained its normal size and presented no sign of fluid, and the patient left the hospital free from pain.

Case LXXXVI. was a good illustration of fluid in the peritoneum resulting from disease of the kidneys. During life the case was regarded as an example of the 'large white kidney,' following nephritis and passing into the fatty kidney, and although one of the kidneys was unexpectedly found very contracted after death from some old disease, you will observe that the other has nearly *four* times the size and weight of a normal kidney.

CASE LXXXVI.—*Fluid in the Peritoneum from Disease of the Kidney—Albuminuria and General Anasarca—Pericarditis and Pleurisy—Death by Uræmia—Great Hypertrophy of Left Kidney and Atrophy of Right.*

James S——, aged 23, was admitted into the Middlesex Hospital under my care, on March 12, 1868. Excepting an attack of 'gastric fever' 2½ years before, his previous health had been good. He had never suffered from scarlet fever; but for nine years he had been in the habit of working in a very hot room, and of drinking gin as well as beer daily. Six weeks before admission, he went from the West End to the City one evening, and got wet through. Three nights after this, on taking off his boots, he noticed

that his feet were swollen, and next morning he found that there was slight swelling of the legs, thighs, trunk, and even of the face. He continued working for two or three days, and then went to St. Bartholomew's Hospital, where he remained a month, but got worse rather than better. Four or five days before coming to the hospital he found his breath becoming short.

On admission, the patient's countenance was extremely anæmic, pasty and puffy. There was considerable œdematous swelling of the trunk, extremities and scrotum. The pulse was 84; the cardiac dulness was slightly increased and the sounds were feeble but otherwise normal. Over the lower fourth of both lungs there was dulness on percussion, with faint, distant, vesicular breathing, and coarse crepitation. The liver and spleen appeared to be of normal size, and there was no jaundice or tenderness of the abdomen, but there was unmistakable evidence of fluid in the peritoneum. The tongue was thickly coated; there was occasional vomiting after food; the bowels were regular. The urine was scanty and smoky, and contained a large quantity of albumen ($\frac{1}{3}$ in volume); it deposited a sediment in which were numerous blood corpuscles and a few granular and oily casts, but no hyaline or epithelial casts. There was slight pain on pressure over the kidneys.

The patient was treated with warm baths and hot air-baths, dry cupping, sinapisms and poultices to the loins; drastic purgatives, such as compound jalap powder, salts and senna, and subsequently elaterium; the perchloride of iron, with large doses of liquor ammoniæ acetatis; and subsequently diuretics, such as the acetate and bitartrate of potash with digitalis.

At first there was slight improvement, but the anasarca and the amount of fluid in the serous cavities gradually increased. From April 1 to April 8, a double pericardial friction-sound was heard over the heart, and on its cessation the cardiac dulness was noted as measuring four inches (instead of two) transversely, and the cardiac sounds were very feeble. On April 8 the urine became almost solid on boiling. On April 11 the dulness had extended over the lower half of both lungs, and there was orthopnoea. The ascites had also increased. On April 11 the patient's countenance became heavy and stupid, his memory was slightly confused, and he was very restless at night and vomited occasionally. On April 13 and 14 the urine was noted as depositing numerous rounded corpuscles distended with oil, about $\frac{1}{800}$ inch in diameter, and very like the compound-granular corpuscles seen in softened brain-tissue. On April 15

there had been no action of the bowels and no urine passed for 24 hours; four ounces of urine drawn off by catheter became almost solid on boiling. The tongue was dry and brown; breath very offensive and the mind confused. On the same evening the patient had a slight attack of convulsions followed by coma, which, notwithstanding sinapisms to the nape and feet, croton oil internally, and hot air-baths, continued until death on the morning of the 17th.

Autopsy.—Brain anæmic; about an ounce of clear serum, containing much urea, in the lateral ventricles and at the base. Nearly a pint of turbid serum in the pericardium. Surface of heart coated with a rough loosely adherent layer of lymph. The heart was large, weighing 19 ounces; there was great hypertrophy of the left ventricle, but the valves were all competent and healthy. Each pleura contained about a pint of clear serum, and the lower lobe of the left lung was coated with a thin layer of recent lymph. The lungs were extremely œdematous. The peritoneum contained several pints of clear serum. The liver, spleen, and mucous membrane of the stomach were all extremely congested. The left kidney was greatly enlarged and weighed $15\frac{1}{4}$ ounces; its surface was smooth and capsule not adherent. The cortex was greatly hypertrophied, measuring at some places $\frac{2}{3}$ inch between the base of a pyramid and the outer surface. The pyramids were congested, but the cortex was pale. The renal tubes were gorged with epithelium cells, most of which were very granular, and at many places they were loaded with oil. Large, globular, compound-granular corpuscles, like those passed in the urine during life, could be seen in the interior of some of the tubes. The right kidney was very small, and weighed only $1\frac{1}{2}$ ounce. Its surface was granular and the capsule adherent. The cortex was dense and atrophied; some of the renal tubes were atrophied; others contained granular or fatty epithelium, or compound-granular bodies. The right ureter was not obstructed nor the pelvis dilated. The right renal artery was pervious, but became suddenly contracted to one-half at about its middle.

Although in the following case, the patient has so far recovered that fortunately no opportunity has as yet been afforded for verifying our diagnosis, there seems little reason to doubt that the fluid in the peri-

toneum was, as in Cases L. and LI. (pp. 260, 263), due to portal obstruction from true cirrhosis of the liver. The liver, however, has not yet undergone much contraction, and this may account for the fact that diuretics and mercury have been so unusually efficacious in removing the fluid from the peritoneum.

CASE LXXXVII.—*History of Spirit-Drinking—Cirrhosis of Liver—Enlarged Spleen—Ascites—Gastro-enteritis—Removal of Ascites by Diuretics.*

Etienne D——, aged 39, a pipe-maker, was admitted into the Middlesex Hospital under my care on April 23, 1868. His mother was alive, aged 76; but his father at 61, and a brother at 28, had died from the effects of hard drinking. The patient himself had for many years been in the habit of drinking largely of beer and spirits, especially the latter, and for the last three years he had been very intemperate, taking large quantities of brandy and rum.

Although he had never had an attack of delirium tremens, he had often been very shaky in the morning. Notwithstanding his habits, he enjoyed good health until three years before admission, when he began to suffer about every week or ten days from severe pinching pains in the abdomen followed by diarrhœa, which was checked by chalk mixture. Nine months before admission there was superadded to this vomiting three or four times a week in the morning, and twelve months before admission he had an attack of bleeding piles. During the last year also his gums had been liable to bleed, and he had been losing flesh. Five weeks before admission the vomiting became more urgent, and he began to suffer from attacks of acute twisting pain in the right side of the abdomen. A fortnight later the vomiting subsided, but diarrhœa came on, and he first noticed his abdomen begin to swell; this swelling rapidly increased and during the last week had caused much dyspnœa. He continued, however, drinking port wine and brandy and water until admission.

The patient's state on admission was as follows:—Body spare,

with sharp features; face sallow; distinct icteric tint of conjunctivæ, but no evident jaundice of trunk or extremities; capillaries of face much enlarged; no puffiness of face or anasarca of extremities. His chief complaints are of great weakness and of swelling and pain in the abdomen, which is not tender, but is much distended and measures 34 inches in girth at the umbilicus. The enlargement is evidently due to fluid in the peritoneum. The abdominal veins are unusually distinct and slightly enlarged, especially in the cœcal region, where they form a distinct network connected with the veins ascending to the chest. The hepatic dulness in the right mammary line appears to be only three inches. In the epigastrium the liver can be felt through the abdominal parietes; it is hard and resisting, without any appreciable nodulation. The splenic dulness is increased, measuring vertically 4 inches and extending forwards to within $2\frac{1}{2}$ inches of the line of the nipple. Tongue coated with a thick yellow fur; appetite indifferent; bowels open about four times a day; motions watery and yellow. Pulse 108; physical signs of heart and lungs normal. Urine acid; sp. gr. 1017; free from albumen, but contains a small quantity of bile-pigment.

The patient was ordered a draught four times a day containing acetate of potash (gr.xx.), spirit of nitric ether (ʒss.), and decoction of broom-tops (ʒjss.); and a pill twice a day with squill (gr.jss.), powder of digitalis (gr.ss.), and blue pill (gr.ijj.). An ointment composed of equal parts of blue ointment and ung. belladonnæ was applied over the abdomen. Alcohol in every shape was interdicted, and the diet was restricted to milk, beef-tea, and farinaceous articles. No attempt was made to check the diarrhœa.

He at once began to improve. He complained frequently of colicky pains in the abdomen and on May 2, 3, 6, and 7, he had attacks of vomiting, and on May 7 slight epistaxis. But from the first the flow of urine increased and the ascites diminished, as shown by the following measurements:—

Girth at umbilicus,	April 23,	34 inches.
”	27,	33·6 ”
”	May 4,	31·75 ”
”	6,	30·75 ”
”	11,	29·25 ”
”	18,	28·5 ”

On May 18 the patient was greatly better. The abdomen was

of the natural size. No evidence of fluid could be detected. The purging and vomiting had ceased; the appetite was improved, and he could eat and retain meat. The splenic dulness was reduced, and the hepatic dulness in the right mammary line measured $3\frac{1}{2}$ inches, the apparent increase in the latter being probably due to its lower margin being no longer obscured by the bowels distended with gas. In the epigastrium the liver could be felt hard and obscurely nodulated.

June 20.—No return of ascites, and is gaining flesh.

Case LXXXVIII. was another example of ascites from chronic atrophy of the liver, which, however, was secondary to valvular disease of the heart (see also p. 274).

CASE LXXXVIII.—*Constriction of Mitral Valve—
Chronic Atrophy of Liver—Ascites and Jaundice.*

Mary T—, aged 61, was admitted into the Middlesex Hospital under my care on February 24, 1868. For twenty years she had been liable to winter cough and for fifteen years she had suffered from palpitations and dyspnœa on exertion. Three years before admission the dyspnœa increased, and after a year the legs began to swell, and she was laid up on this account for three months. After another year she first noticed swelling of the abdomen, which, as well as the swelling of the legs, increased slowly. She stated that she had never had rheumatic fever, but that for several years she had suffered from pains in her limbs and also from attacks of diarrhœa. The catamenia had ceased at the age of 30. Several weeks before admission her skin became yellow.

On admission, the patient's countenance was anxious; her lips and cheeks were livid; there was great œdema of both lower extremities with tension of the integuments. There was also considerable œdema of the left arm, but no puffiness of the face or swelling of the right arm. The abdomen was greatly distended with fluid in the peritoneum, measuring at the umbilicus 39 inches. The pulse was 120, small and feeble, but regular. The apex of the heart beat between the fifth and sixth ribs, slightly to the left of the left nipple. The transverse cardiac dulness was increased, measuring three inches, the increase being mainly to the left. Over

the point of impulse a prolonged pre-systolic bellows-murmur was audible. The respirations were 40 and embarrassed. There was frequent cough, with expectoration of puriform mucus. Bronchitic râles were audible over the greater part of both lungs and over the lower half of both posteriorly, there was coarse crepitation and feeble breathing. The tongue was moist and furred. The patient had great thirst and no appetite; she vomited frequently after food, and complained of tenderness at the epigastrium. The bowels had been open three or four times in the night before admission. The hepatic dulness was much diminished, not exceeding two inches in the right mammary line. There was a decided icteric tinge of the skin and conjunctivæ, and a distinct reaction of bile-pigment in the urine, which was scanty, but contained no albumen. The patient's mind was clear, but she slept very badly, owing to the orthopnoea.

The patient was treated with diuretics (nitric ether, acetate of potash, and decoction of broom-tops, with a pill of digitalis, blue pill, and squill) and stimulants (gin 8 ounces and brandy 4 ounces), while mustard and linseed poultices were applied over the chest. The prostration and dyspnoea nevertheless increased. On February 26, the pulse was 86, extremely feeble and irregular. The respirations were 48, but interrupted by frequent cough; the expectoration consisted of nummular masses of deep yellow pus. The veins of the neck were turgid with slight regurgitation from below. The bowels had acted six or seven times in the night. The dropsy had increased, and the right hand and arm were now œdematous. These symptoms continued until the patient's death at midnight between February 27 and 28.

Autopsy.—The heart weighed 14 ounces. The mitral orifice was much constricted, just admitting the tip of the finger, and the flaps of the valve were very rigid and thickened from fibrous and calcareous deposit. There was also calcareous deposit in the aortic valves, which, however, were competent. The tricuspid orifice was slightly dilated. There were firm adhesions between the opposed surfaces of both pleuræ; the pulmonary pleuræ were thickened; and the lower part of both lungs was condensed from intersecting fibrous bands, with œdema and congestion of the intervening pulmonary tissue. The bronchial tubes were dilated, their walls thickened, and their interior filled with pus. The peritoneum contained several quarts of yellow serum. The liver was very small and dense, with much fibrous deposit on its surface and also extending into its

interior. The outer surface was at several places granular, the depressions corresponding to the centre of the lobules. The liver weighed only 31 ounces.

The mucous membrane of the stomach was intensely injected, with much adherent mucus and numerous hæmorrhagic erosions. The kidneys were slightly granular.

In the last case which I shall bring before you, the cause of the fluid in the peritoneum was somewhat obscure. The concurrence of urgent diarrhœa with rapidly increasing ascites pointed to some obstruction of the trunk of the portal vein; while the enlargement of the liver, the great enlargement of the spleen, the large quantity of albumen in the urine without general dropsy, the great anæmia, and even the effects of treatment, which in its success far exceeded what at first was anticipated, made it probable that the compression of the portal vein was due to lymphatic glands enlarged from waxy deposit.

CASE LXXXIX.—*Great Ascites and Diarrhœa—Enlargement of the Spleen and Liver and Albuminuria.*

Sarah B—, aged 25, a respectable married woman, was admitted into the Middlesex Hospital under my care on April 21, 1868. Her father died at the age of 40 from the effects of an accident. Her mother died at 36 from what the patient believed to have been cancer of the womb, but for a long time she had suffered from cough and expectoration. The patient herself was one of a family of five; one brother had died at 21 of rapid consumption; the remainder were healthy. The patient stated that the only serious illness from which she had suffered was when the catamenia appeared at the age of 12. The discharge was profuse and recurred at irregular intervals, so that she was confined to bed for three months from debility. Since then the catamenia had been regular but always profuse, and she had suffered from great weakness and

depression of spirits, for which she had frequently been an out patient at different hospitals. Two years ago she was married, and since then her health had been worse, but she had never been pregnant. For twelve months she had suffered frequently from pain in her right side.

The illness for which she came to the hospital commenced 10 weeks before admission with swelling in the abdomen which gradually increased, and latterly had been the seat of considerable pain, especially when she was up. There had been neither vomiting nor jaundice, but from the first the bowels had been very relaxed, at least five or six times a day, and for the last three or four days as often as ten or twelve times; the motions had been dark and very offensive. The patient had lost much flesh, but had not suffered from cough, rigors, or perspirations. The legs had swollen occasionally after walking.

The patient's state on admission was as follows:—Body emaciated; extreme debility. Countenance sallow and chlorotic, but conjunctivæ perfectly white. No puffiness of face, and only very slight pitting about ankles. The abdomen greatly enlarged, very tense, and presenting all the signs of a large quantity of fluid in the peritoneum. Girth at umbilicus $34\frac{1}{2}$ inches. Considerable enlargement of the abdominal veins. The upper margin of the liver is on a level with the nipple and in the right mammary line measures $4\frac{1}{4}$ inches. Area of splenic dulness much increased, measuring vertically about 6 inches, and the organ can be felt projecting beyond the ribs to $1\frac{1}{2}$ inch in front of the left mammary line. Complaints of considerable pain in abdomen, apparently due to the distension; no marked tenderness, and tolerably free movement of abdominal walls. Pulse 96; apex of heart elevated to between fourth and fifth ribs, immediately to the right of the nipple; cardiac dulness not increased and sounds normal. No cough; respirations 30; physical signs of lungs normal. Tongue moist, slightly furred; gums anæmic; breath offensive; appetite moderate; no retching; motions watery and yellow. Urine high-coloured; sp. gr. 1024; contains a large quantity ($\frac{1}{3}$ in volume) of albumen, but no casts of the uriniferous tubes. White corpuscles in blood not increased. Temperature 98° .

The patient was ordered an astringent draught every four hours of tincture of catechu (ʒj.) and laudanum (ʒv.); meat diet and 4 ounces of brandy. The diarrhœa, however, continued, the bowels being open about twelve times a day. On April 24 pills with

acetate of lead (gr. iij) and opium ($\frac{1}{3}$) were substituted for the astringent draught, and milk and arrowroot for the meat, but without effect. On April 25 the patient had still about twelve motions a day; she had lost her appetite and was much weaker. The brandy was increased to 6 ounces, and a draught containing tannine (gr.x.), tincture of opium (℥v.), glycerine (3ss.) and aq. menthæ (3j.), was ordered to be taken every four hours. This had the effect of diminishing the diarrhœa, and on May 4 it was noted that the patient's appearance was improved, that there were only three motions a day, and that the quantity of albumen in the urine was greatly diminished ($\frac{1}{20}$), but that the girth at the umbilicus was slightly increased ($34\frac{1}{2}$ inches). The catamenia appeared on April 24, a week before their time, and lasted till April 28, and were rather abundant. The patient was now ordered a draught four times a day containing nitro-hydrochloric acid (℥x.), tincture of opium (℥v.), syrup (3ss.), and water (3j.).

With the exception of flatulent pains, which were relieved by vegetable charcoal, the patient from this time continued to improve steadily, and on May 18 her condition was noted to be as follows: Looks and feels better. Has been out in the garden for the last three days. Bowels open only once a day. Urine contains only about $\frac{1}{20}$ of albumen. The abdomen is lax and measures 33 inches at the umbilicus; the signs of fluid have almost disappeared, and the enlargement appears due mainly to gas in the bowels. Judging by her clothes, the abdomen seems five or six inches smaller than on admission.

On June 2 the patient left the hospital, her abdomen measuring only 32 inches, and presenting no signs of fluid.

LECTURE XII.

A. PAIN IN THE LIVER—B. GALL-STONES—C. ENLARGEMENTS OF GALL-BLADDER.

A. HEPATIC PAIN SIMULATED BY: 1. PLEURODYNIA — 2. INTERCOSTAL NEURALGIA — 3. PLEURISY — 4. GASTRIC DYSPEPSIA — 5. INTESTINAL COLIC — 6. RENAL COLIC. THE VARIETIES AND CAUSES OF GENUINE HEPATIC PAIN.

B. GALL-STONES. THEIR VARIOUS CONSEQUENCES AND SYMPTOMS.

C. ENLARGEMENT OF GALL-BLADDER. ITS CAUSES, CLINICAL CHARACTERS, AND TREATMENT.

A. HEPATIC PAIN.

GENTLEMEN,—Pain is sometimes the most prominent symptom of disease of the liver, and is often an important aid to diagnosis. You must, however, beware of being misled by patients, who constantly ascribe pain to the liver with which that organ is in no way concerned. In forming a diagnosis you must keep constantly in view the various conditions which may simulate hepatic pain. They are mainly :—1. Pleurodynia, 2. Intercostal Neuralgia, 3. Pleurisy, 4. Gastric Dyspepsia, 5. Intestinal Colic, 6. Renal Colic.

1. *Pleurodynia*, or rheumatism of the intercostal muscles, may be situated in the right hypochondrium, and then the acute pain, increased by pressure, by movement, by taking a long inspiration, or by cough-

ing, and accompanied by short jerking respirations, may be mistaken for the pain of peri-hepatitis; but it differs from this—

a. In the pain being more localized. It is often confined to one spot between two ribs, and there is no tenderness on pressure over the epigastrium, or elsewhere in the hepatic region than in the spot to which the patient refers the pain.

b. In the absence of symptoms of pyrexia or of constitutional disturbance.

c. In the absence of any other symptom or sign of hepatic disease.

d. In the occasional coexistence of muscular rheumatism in other parts of the body.

2. *Intercostal Neuralgia*, except that the pain is more intermittent, may present many of the characters of pleurodynia, and may like it be localized in the hepatic region; but on the whole it is chiefly met with in the sixth to the ninth intercostal spaces on the left side, and in females. When it occurs in the hepatic region, it differs from true hepatic pain—

a. In the pain being chiefly referred to three points in the course of the nerve, viz. in the vertebral groove, in the axillary region, and at the termination of the nerve in front.

b. In the frequent coexistence of neuralgia of the mammary gland, spinal irritation, or cutaneous hyperæsthesia.

c. In the absence of any other symptom or sign of hepatic disease.

3. *Pleurisy* may give rise to a pain, which, like that of pleurodynia, is increased by pressure, movement, inspiration, or coughing, but which differs in being associated with more or less pyrexia, and if the inflammation be at the base of the right pleura, the pain may be indistinguishable from that of peri-hepatitis. It is probable indeed that in some of the cases of so-called 'diaphragmatic pleurisy' the inflammation is on the under surface of the diaphragm rather than on the upper, while observations in the dead house leave little doubt that in many there is inflammation on both sides. Dulness on percussion or pleuritic friction over the base of the lung, or the concurrence of pneumonia, will often assist the diagnosis in favour of pleurisy; but in slight cases of diaphragmatic pleurisy there may be neither dulness nor friction, and peri-hepatitis will occasionally give rise to a rubbing sound during the respiratory movements. (See Case VII. p. 87.)

4. *Gastric Dyspepsia*. Patients very commonly refer the pain arising from various disorders of the stomach to disease of the liver. The liver is said to be 'out of order,' when the stomach or duodenum is the organ really at fault.

a. Pain coming on after food may be due to derangement of the stomach or duodenum, or more rarely to congestion of the liver, but the liver is not likely to be the source of the pain, unless there be tenderness on pressure in the right hypochondrium, and those other signs of congestion of the liver which I have already described to you (p. 120).

b. Attacks of severe pain (gastrodynia) occur in the stomach independently of food, and may simulate hepatic colic, or hepatic neuralgia. From hepatic colic it will be distinguished by its situation, by its being often accompanied by the eructation of watery fluid (pyrosis), and by the absence of jaundice. Neuralgia of the stomach can only differ from hepatic neuralgia in the situation of the pain.

5. *Intestinal Colic* resembles hepatic colic in there being paroxysms of severe abdominal pain with vomiting, but without tenderness on pressure or fever, but differs from it in—

a. The situation of the pain, which is referred to the umbilicus rather than to the gall-bladder and right scapula.

b. The absence of jaundice.

c. The circumstances under which it occurs, viz. : constipation, some obvious error in diet, or the presence of lead in the system, indicated by the blue line along the margin of the gums or by a history of lead colic or palsy.

6. In *Renal Colic* there is also severe paroxysmal abdominal pain with vomiting and without fever, but—

a. The pain is referred chiefly to one kidney, which is often tender, and thence it shoots down the corresponding thigh and down to the corresponding testicle, which is retracted.

b. There is no jaundice.

c. The urine contains blood, or there is a previous

history of hæmaturia, or of the passage of a calculus by the urethra.

Keeping in mind these sources of fallacy, we may proceed to consider the varieties and causes of pain which is justly referable to the liver. It will suffice in most instances if I merely mention the diseases in which the pain occurs, inasmuch as I have already described their leading characters to you in former lectures. I may remind you, however, that certain diseases of the liver are characterized by a remarkable immunity from pain, and more especially the waxy liver (p. 27), the fatty liver (p. 45), simple hypertrophy (p. 52) and atrophy (p. 221), and hydatid tumour (p. 58).

Pain having its source in the liver may be said to present three varieties.

I. First, there is a pain which is very severe, comes on in paroxysms with distinct intermissions, and is associated with little or no tenderness and with no fever, but is often attended or followed by jaundice. Pain answering to this description results from :

1. The presence of gall-stones or other foreign bodies in the bile-ducts (see pp. 319, 325, and 327).

2. Obstruction of the common bile-duct by a duodenal ulcer (see p. 334).

3. An aneurism of the hepatic artery (see p. 345).

4. Hepatic neuralgia. This has been described by Andral, Frerichs,* Budd,† and other writers, and

* Frerichs, *Dis. of Liver*, Syd. Soc. Ed., ii. 548.

† Budd, *Dis. of Liver*, 3rd ed. p. 380.

although it is probable that in many of the reported examples, and especially in those where there has been jaundice, the pain has been due to gall-stones which have escaped observation, there are others which, occurring in nervous persons or hysterical females, often at tolerably regular intervals of about a month, associated with other nervous symptoms and with no jaundice, seem really to be instances of neuralgia of the hepatic plexus of nerves. During the last five years I have had under my observation a case of this sort, the particulars of which I shall relate to you immediately (Case XC.).

II. Secondly, there is a pain in the liver which is not severe and is often described rather as a feeling of weight or distension, which is often associated with pain in the right shoulder, which does not intermit, and which is slightly increased by pressure, or by lying on the left side,* or after meals. This pain is usually associated with slight febrile disturbance and more or less jaundice. Pain of this description is observed in:—

1. The various forms of congestion of the liver (p. 121).
2. The early stages of hepatitis (p. 169).
3. Catarrh of the bile-ducts (p. 132).
4. Obstruction of the common duct followed by great accumulation of bile within the liver (p. 144).
5. The pain in acute atrophy (p. 227) partakes somewhat of this character.

* Great enlargement from any cause will also give rise to a dragging pain, when the patient lies on the left side.

III. A third form of pain is constant and severe, is greatly aggravated by pressure, movement, or coughing, and is associated with more or less fever, but not necessarily with jaundice. This is the pain of perihepatitis, which, as I have already told you, although sometimes a primary and independent affection, is more commonly secondary to other diseases of the liver, and thus acute pain may occur in diseases such as cirrhosis (p. 249), waxy enlargement (p. 27), or hydatid of the liver (p. 58), whose natural course is painless, or may aggravate the already existing pain of cancer (p. 190). Whatever may be the primary disease of the liver, pain of the character last described always indicates inflammation of the capsule, and its supervention sometimes furnishes indications of some importance. Its occurrence, for instance, in a case of hydatid of the liver would indicate that the hydatid was about to burst or contract adhesions to some of the adjacent viscera.

In former lectures I have brought under your notice many examples of different diseases of the liver in which pain was a prominent symptom, and the only case which I shall now relate is the one of hepatic neuralgia already referred to.

CASE XC.—*Hepatic Neuralgia.*

Charles B——, aged 20, a banker's clerk, came to me on July 11, 1863, complaining of a pain in the hepatic region, which came on in paroxysms, about once a month. He would go to bed quite

well at night, but during the night, or in the morning, he would awake with pain below the right ribs, shooting back to the scapula, and so severe as to make him bend himself double, and prevent him going to his business. This pain would last all day, without remissions or exacerbations, but in the following night it would go away almost as suddenly as it had come, and next day he would go to business as usual, feeling only slight languor. The pain was usually accompanied by tenderness and a sense of fulness and tightness in the region of the liver, by nausea, and occasionally by vomiting, but not by headache, and, on no occasion, either during or after the attacks, had there been jaundice. The patient was unable to assign any cause for the attacks, to which he had been subject for several years since coming from Scotland to London; they could not be traced to errors in diet, and in the intervals of the attacks he did not suffer from symptoms of indigestion. The liver and gall-bladder were very carefully examined, but nothing abnormal could be discovered in their size or shape, and in the intervals of the attacks there was not the slightest tenderness in the right hypochondrium. All the other organs of the body also appeared healthy; the characters of the urine were natural, and the bowels were regular. The patient appeared to be of a somewhat nervous disposition, and his spirits were depressed owing to the frequent paroxysms of pain interfering with his business.

Various remedies were employed with the object of relieving or arresting the paroxysms of pain, such as mustard poultices, purgative pills of calomel, colocynth and henbane, emetics, pills of opium and cajeput oil, and warm baths; but no marked effect was observed from any of them. Under the use, however, of tonics, such as the nitromuriatic acid with infusion of gentian, quinine, and iron, the patient felt stronger, and in better spirits, and the intervals between the attacks of pain became longer. The attacks, however, still recurred about once in six weeks or two months, until January, 1866, when, apparently from sitting during several days in succession with wet boots after walking in snow, he was seized with symptoms of pleurisy in the left side, which confined him to bed for three weeks. After this he returned to his business, but in the following May, he was attacked with copious hæmoptysis, and the hæmorrhage recurred several times during the summer. He had at that time all the signs of tubercle in the upper part of the left lung, and there is still (June 1868) dulness below the left clavicle. Since the autumn, however, of

1866, he has been able to follow his occupation; he has had no return of the hæmoptysis; he has no cough and feels well; and, what is more remarkable, since the attack of inflammation in the chest in January, 1866, there has been no recurrence of the paroxysms of pain in the right hypochondrium.

B. PATHOLOGICAL CONSEQUENCES OF GALL-STONES.

In what remains of this lecture I wish to say a few words on the subject of gall-stones and diseases of the gall-bladder. In a former lecture, when speaking of the causes of jaundice, I described to you at some length the consequences of gall-stones passing along the bile-ducts, or becoming impacted in the common duct, but in doing so I by no means exhausted their clinical history. I purpose now supplying this deficiency by calling your attention to the different situations in which gall-stones are found, to the erratic courses which they sometimes take, in their endeavour, so to speak, to escape from the body, and to the varying symptoms and dangers which may result from them accordingly.* Those of you who wish to have further information on the subject of gall-stones, I would refer to the elaborate Memoir of M. Fauconneau-Dufresne which obtained the prize of 1500 francs from the French Academy of Medicine in 1847.

1. *Gall-stones may be retained in the gall-bladder.*—The gall-bladder is the portion of the biliary passages in which calculi are found most frequently and in

* La Bile et ses Maladies, Mém. de l'Acad. Roy. de Méd. 1847, xiii. p. 36.

largest quantity, and there is abundant evidence that they may exist there for a long time, without giving rise to symptoms of any sort. You will constantly find concretions in the gall-bladder after death in the bodies of persons who during life have exhibited no symptoms of their presence. But occasionally when the concretions are numerous and large, they cause a sensation of uneasiness, weight, tension, or dragging, in the region of the gall-bladder, which is usually worse after meals, after any violent muscular exertion, or after driving over rough roads. I have now under my care a lady who has suffered from gall-stones, and who frequently complains of a sensation of a heavy weight rolling from side to side in the situation of the gall-bladder when she turns in bed. Fauconneau-Dufresne quotes a precisely similar case from Fabricius Hildanus.* Gall-stones in the gall-bladder also now and then cause vomiting and other derangements of the stomach, and their pressure on the stomach has been known to determine all the symptoms of stricture of the pylorus. It is very possible also that in persons of a nervous constitution they may be a centre of irritation, from which may arise uneasy sensations and symptoms of actual disease in distant parts of the body, with great mental depression and hypochondriasis. Several cases which have been under my care have served to impress me strongly with this opinion.

When the gall-bladder is full of concretions, it

* Fauconneau-Dufresne, *op. cit.*, p. 274.

sometimes forms a tumour which is appreciable through the abdominal parietes, and the real nature of which may be recognized by its hard and resisting character. On palpation, also, a peculiar crackling sensation, or an actual sound, is sometimes elicited, which has been aptly compared to that produced by grasping a bag of hazel-nuts, or by rolling about small pebbles in the mouth. The stethoscope in these cases may afford material assistance in the diagnosis.

Gall-stones retained in the gall-bladder may also excite inflammation and ulceration of the mucous membrane and ulterior consequences, to be presently referred to.

2. *Gall-stones may become impacted in the neck, or in the cystic duct, of the gall-bladder.*—When a concretion passes from the gall-bladder into the cystic duct, it usually gives rise to vomiting and the symptoms of biliary colic already described (p. 319), but as long as it does not advance beyond the cystic duct there is no jaundice. Sometimes the calculus never reaches the common duct; it becomes impacted in the cystic duct, or it drops back into the gall-bladder, and in either case the colic may cease without there having been any jaundice. At the same time in post-mortem examinations, one occasionally finds the neck of the gall-bladder obstructed by an impacted gall-stone, although no symptom leading to the suspicion of gall-stones has ever been observed during life. Permanent obstruction of the cystic duct by a calculus occasionally leads to inflammatory enlarge-

ment of the gall-bladder, to the consideration of which we shall presently return.

3. *Gall-stones may form in the radicles of the hepatic duct in the interior of the liver.*—It is not often that concretions form in the bile-passages within the liver, inasmuch as the bile is not here subjected to those conditions of concentration and repose which contribute so much to their formation in the gall-bladder. They have frequently been found, however, in the dilated bile-ducts within the liver in cases of obstruction of the ductus communis choledochus, and there are also a few cases on record where biliary concretions have been found within the liver independently of any such obstruction. These concretions are often numerous, but very small, constituting what has been called ‘biliary gravel;’ at other times they are large and branched like a piece of coral, as in this drawing, which is copied from one of Cruveilhier’s Pathological Plates.* Chopart met with a case where the liver contained so many concretions that it could not be cut with a scalpel.† These concretions in the liver may induce partial obstructions of bile and dilatation of the ducts; sometimes they become enclosed in firm fibrous cysts, shut off from the bile-ducts; and at other times they cause ulceration of the ducts and multiple abscesses in the liver.†

The symptoms of intra-hepatic concretions are usually obscure. They do not cause jaundice or enlargement of the liver and the smaller concretions

* Livraison xii. Pl. V. † Fauconneau-Dufresne, op. cit., p. 249.

may cause no pain. But now and then they give rise to a feeling of weight, or of dull pain, in the region of the liver, with sudden attacks of sharp cutting pain or violent colic shooting from the right hypochondrium through the chest or to the hypogastrium, while in other cases they have occasioned attacks of rigors,* followed by heat and sweating, which have simulated ague. Fauconneau-Dufresne records at length a case which was diagnosed by Professor Trousseau, from his finding biliary concretions in the motions associated with attacks of pain like that which I have described.†

4. *Gall-stones may be lodged in the hepatic duct, before its junction with the cystic duct*, but this is a rare occurrence. A concretion in this situation must be derived from the ducts within the liver, and if it has succeeded in escaping from the small ducts in the liver, it is not likely to encounter any serious obstacle to its progress, or to cause symptoms of any importance in its passage through the larger hepatic duct. It is not surprising then that there are very few cases on record where a large gall-stone has been found obstructing the hepatic duct after death; but where this does happen during life, there would be jaundice, enlargement of the liver, vomiting, biliary colic, and those other symptoms of obstruction of the common duct which have been already detailed to you (p. 142). There would, however, be no enlargement of the gall-bladder, as when the common duct is obstructed.

* Frerichs, op. cit., ii. 516.

† Op. cit., p. 270.

5. *Gall-stones may be lodged in the ductus communis choledochus.*—This, in fact, is one of their most common situations, and they find their way here from one of two sources, either from the intra-hepatic ducts, or more commonly from the gall-bladder. As a rule they are discharged sooner or later into the duodenum, their passage being marked by those symptoms of biliary colic which I have already endeavoured to describe to you (p. 319). The passage of the concretion through the cystic duct gives rise to severe colic, but when it enters the common duct, jaundice is added to the former symptoms, and, in consequence of the larger size of the common duct, the pain usually abates in severity, again to return with even increased intensity on the arrival of the concretion at the narrow duodenal orifice, on dropping through which it sometimes ceases suddenly, as if by enchantment. More rarely the calculus is firmly impacted in the common duct and becomes one of the causes of permanent jaundice. A rough angular stone will experience more difficulty, and cause more pain, in passing along the duct, than one which may be larger but is rounded and smooth, but at the same time it is more likely to permit a little bile to trickle past it. Now and then the common bile-duct is found dilated into a large pouch containing numerous calculi, but at the same time allowing the bile to pass on to the bowel. I recently met with this condition in the body of a lady, whose case I shall take another opportunity of relating to you (Case XCI.),

and a similar appearance is figured in one of Cruveilhier's Plates.* Morgagni long ago reported a case where the gall-bladder was dilated to the size of the stomach and was full of calculi,† and more recently a similar case has been recorded by Frerichs.‡

6. *Gall-stones may excite inflammation and ulceration of the lining membrane of the gall-bladder or bile-ducts, and may thus lead to perforation and peritonitis or pyæmia.*—While still in the gall-bladder or in any part of the biliary passages, gall-stones may be followed by other consequences besides those already mentioned. Their pressure or irritation may excite inflammation of the mucous membrane with which they are in contact, and this may spread through the entire biliary passages. You will remember that, when lecturing on inflammation of the biliary passages, I referred to the irritation of gall-stones as one of its causes (p. 134), and that not long ago we had an instance of inflammation from this cause in the wards (Case XXIX., p. 137). Occasionally, and more especially when the cystic duct is obstructed, inflammatory products accumulate in large quantity in the gall-bladder, from which all the colouring matter of the bile is absorbed, and the gall-bladder becomes converted into a large, painful, fluctuating tumour, simulating an abscess, which may burst in different directions, or may be opened by the

* Anat. Path. Livraison xxix. Pl. IV. fig. 3.

† Trousseau, Clin. Méd. ii. 539.

‡ Op. cit., ii. 469.

surgeon, as in Case XCIII. The pressure of gall-stones may also cause ulceration, which may not be limited to the mucous membrane, but may perforate the entire coats of the gall-bladder or a bile-duct, and induce fatal peritonitis from the escape of bile or of the concretions themselves into the peritoneum. I show you here some gall-stones removed from the body of a lady lately under my care who died suddenly of peritonitis from ulcerations and perforation of the gall-bladder (see Case XCI.), and you will find in Fauconneau-Dufresne's *Memoir* an observation quoted from Dr. Wolf, where death from peritonitis was due to complete rupture of the hepatic duct.* Trousseau also mentions a case where a calculus and a considerable quantity of bile escaped into the peritoneum through a rupture in the ductus communis choledochus.† In some of these cases the rupture seems to be independent of ulceration, but to have been the result merely of undue pressure upon the coats of the biliary passages, which have perhaps been weakened by inflammation or fatty degeneration.‡ Fatal peritonitis in these cases is sometimes prevented by the formation of adhesions and the establishment of biliary fistulæ, or the calculus may escape from the gall-bladder and become encysted in its vicinity by organized lymph.§ Cases have also been recorded

* *Op. cit.*, p. 273.

† *Clin. Méd.* ii. 533.

‡ See Budd, *op. cit.*, p. 234.

§ For example, see Simon, *Path. Trans.* v. 157, and Sharman, *Med. Times and Gazette*, 1859, i. 274.

where gall-stones have escaped by ulceration from the biliary passages into the substance of the liver, and been there found in the interior of abscesses which communicated with the biliary passages.* More commonly it happens that the ulceration or sloughing produced by the pressure or irritation of the gall-stones contaminates the portal blood, and leads to the development of multiple pyæmic abscesses in the manner already explained to you. I would recall to your recollection, by way of illustration, the instructive case of Mrs. — (Case XXXV., p. 158), which was related to you in a former lecture. You will thus see that an attack, which in the first instance may appear to be nothing more than one of colic from gall-stones, may suddenly and unexpectedly assume all the symptoms of peritonitis or pyæmia, and prove fatal; and the practitioner, if he be not on his guard in giving a prognosis, may incur unmerited discredit.

7. *A gall-stone which has reached the bowel may be voided per anum.*—Once arrived in the bowel the natural exit of a gall-stone from the body is by the anus, and this is the termination which it is the great object of treatment to promote. As a rule all symptoms cease as soon as the concretion leaves the bile-duct, but occasionally when the concretions are large, or when numerous concretions become cemented together by faecal matter into one large mass, their discharge per anum is preceded by severe colic, vomiting, and great

* Fauconneau-Dufresne, op. cit., p. 340.

prostration. Biliary calculi voided per anum are sometimes remarkable for their size. Fauconneau-Dufresne* and Frerichs† have collected the notes of several cases where they were as large as a pigeon's, or even a hen's egg, and in the Museum of the Royal College of Surgeons are two calculi passed per anum, one of which measures $1\frac{3}{4}$ inch by $1\frac{1}{2}$ inch, and the other is nearly 2 inches in length.‡ During the present session also of the Pathological Society two specimens of oval biliary concretions passed per anum whose long and short diameters measured about $2\frac{1}{4}$ inches by $1\frac{1}{2}$ inch, were exhibited by Dr. Hilton Fagge. In most instances of these large concretions it is probable that they have found their way into the bowel, not through the bile-duct, but through a biliary fistula from the gall-bladder, although Rokitsansky observes that owing to the extreme distension which the biliary passages are capable of, calculi of the size of a hen's egg are enabled to pass through them.§

8. *Gall-stones may be vomited from the stomach.*—In rare instances gall-stones are expelled from the body by vomiting. Two instances are reported by J. L. Petit, in one of which the calculus was $2\frac{1}{2}$ inches long.|| Eight others have been collected by Fauconneau-Dufresne;¶ one is reported in the Pathological Transactions by Mr. Jeaffreson of Framlingham, where the

* Fauconneau-Dufresne, op. cit., p. 319.

† Frerichs, op. cit., ii. 523.

‡ Catalogue of Calculi, pp. 168, 176.

§ Path. Anat. Syd. Soc. Transl. ii. 165.

|| Mém. de l'Acad. Roy. de Chir., 1743, vol. i. p. 308.

¶ Op. cit., p. 306.

concretion was larger than a nutmeg;* and one by Dr. E. J. Miles, where sarcinous vomiting ceased on the ejection from the stomach of two large gall-stones.† The expulsion of the stones has usually been preceded for several days by severe pain in the stomach, and accompanied by violent and protracted vomiting. It has been the custom to account for these cases on the supposition of an antiperistaltic action of the duodenum, but in most of the recorded instances it is probable that the calculi have found their way into the stomach by a direct fistular communication with the gall-bladder. This view is favoured by no mention being made of jaundice in many of the cases; by several calculi being vomited in some,—in one as many as twenty; and by one patient vomiting a calculus on three different occasions at intervals of several years.‡ The possibility, indeed, of a large calculus passing backwards through the pylorus is very doubtful.

9. *Gall-stones after entering the bowel may be impacted and become a cause of intestinal obstruction.*—I show you here a specimen consisting of a portion of the ileum with a large gall-stone tightly fitted into its interior like a cork, which was obtained from the body of a patient who died in this hospital a few years ago with all the symptoms of intestinal obstruction

* Vol. xii. p. 129.

† Lancet, Jan. 19, 1861.

‡ Since this lecture was delivered, the sequel of Mr. Jeaffreson's case has been published by his brother, Dr. H. Jeaffreson, from which it would appear that the patient died shortly after the report of her case to the Pathological Society, and that the gall-stone was found to have escaped by ulceration into the duodenum. Brit. Med. Journ. May 30, 1868.

(Case XCII.), and medical literature contains the records of many similar cases.* The intestine in these cases is greatly distended above the obstruction, but contracted and empty below. The impaction of the calculus in the small intestine is marked by obstinate constipation, vomiting, first of food, then of bile, and lastly of stercoraceous matter, pain and tenderness of the abdomen and other symptoms of peritonitis, which symptoms continue until death, or until the concretion escapes into the large bowel. Although these cases are often fatal, it is satisfactory to know that in several of the 20 cases of which I have collected the notes the concretion has been discharged per anum, and the patient has recovered, even after stercoraceous vomiting had set in. In one of the cases (Dr. Omond's) stercoraceous vomiting had lasted upwards of three weeks, and yet the patient recovered.

A concretion which finds its way through the bile-duct is not likely to become arrested in the bowel. Accordingly, in most cases of obstruction of the bowel by a biliary calculus, this has passed directly from the gall-bladder into the bowel by a fistulous opening, and

* Five cases collected by Fauconneau-Dufresne, *op. cit.*, p. 311; two observed by Cruveilhier, *Traité d'Anat. Path.* ii. 543; two by Frerichs, *op. cit.*, ii. 524; one by Oppolzer, *Zeitsch. d. Gesellschaft d. Aertze in Wien*, Nov. 1860; one by Sir Thos. Watson, *Lect. Pract. Phys.* 3rd ed. ii. 465, which is the same as Mayo's case included in Fauconneau-Dufresne's five; one by Dr. Omond, *Lond. Med. and Surg. Journ.* 1836; one by Peacock, *Trans. Path. Soc.* i. 255; one by Pye Smith, *ib.* v. 163; one by Baly, *ib.* x. 185; one by Potts, *ib.* xv. 105; one by Dr. Ezra Palmer, *Records Med. Soc. Bost. U.S.*, vol. iii. 106; one in *Coll. Surg. Mus. Path. Ser. No. 1182*; one in *Barth. Hosp. Mus. Ser. xvi. No. 84*. See also the references to Cystico-duodenal fistulæ, p. 514.

thus there may be no previous history of jaundice, to assist in diagnosing the cause of the obstruction. For example, this was what had occurred in the case of which I have shown you the specimen. On the other hand, you will remember Rokitansky's statement that a calculus as large as a hen's egg may pass through the bile-ducts. Abercrombie also relates the case of a man who after many attacks of colic followed by jaundice, died on the fifth day of a similar attack, with the addition of symptoms of ileus. A biliary calculus, measuring 4 by $3\frac{1}{2}$ inches in circumference, was found plugging the ileum; the gall-bladder is spoken of as inflamed and partially disorganized, but no mention is made of a fistula, while the common duct was patent and easily admitted the finger.* In another case, reported in the *Pathological Transactions* (XV. 106), where the concretion in the ileum had a circumference of four inches, there were no adhesions about the gall-bladder. It is possible that a biliary concretion may acquire increased dimensions in the bowel from the deposit of fæcal matter, and sometimes they form the nucleus of large intestinal concretions.† It is right to add that several instances have been observed where enormous biliary calculi were lodged in pouches of the duodenum or jejunum, without causing obstruction of the bowels.‡

Several cases have been recorded where the large

* *Diseases of Stomach*, 3rd ed., p. 127.

† See Dr. P. H. Watson, *Ed. Med. Journ.*, May 1868, p. 989.

‡ See Duffin, *Lancet*, May 27, 1848 and G. Harley, *Path. Trans.*, viii.

intestine has been obstructed, just above the sphincter ani by a large biliary concretion, or by several concretions cemented together by faecal matter. Obstinate constipation and considerable pain have been the result, but under suitable treatment the concretion has generally been voided per anum and the patient has recovered.

10. *Gall-stones may excite ulceration or gangrene of the bowel, and may even escape from it by a perforation into the peritoneum or externally.*—In obstruction of the small intestine by biliary concretions, death usually occurs before there is time for perforation. Perforation is most apt to happen when the concretion is situated in a portion of the bowel, where it can remain for a long time without causing obstruction. Thus several instances have been noted where biliary concretions in the cœcum have caused ulceration, gangrene, perforation, and fatal peritonitis. In rare cases gall-stones have been known to enter the appendix vermiformis, and, like other foreign bodies, or concretions formed in the part, to cause ulceration, perforation, and fatal peritonitis. In most of the cases where biliary concretions either in the cœcum or in the appendix have been recorded as producing perforation, it may be doubted if the concretions were not intestinal rather than biliary.* Budd, however, observed a case where a gall-stone in the vermiform appendix caused perforation and fatal peritonitis,† and

* This remark, I suspect, applies to most, if not all, of the cases collected by Fauconneau-Dufresne. Op. cit., pp. 313, 316.

† Dis. of Liver, 3rd ed., p. 378.

another patient died from the same cause under the care of Trousseau.* The specimen of a similar case is preserved in the Museum of St. Bartholomew's Hospital; † and some years ago a case was related by Dr. Adolphe Sirey where a circumscribed abscess formed round the appendix, and where the concretion ultimately found its way out by an ulcerated opening through the abdominal wall.‡ Lastly, Dr. Horace Jeaffreson has quite recently recorded an interesting case where two large gall-stones became shelved in a slightly pouched portion of the ileum just above the valve, and eventually set up irritation and ulceration, which resulted in perforation and fatal peritonitis, and where one of the gall-stones was found to have escaped into the peritoneum.§

11. *Gall-stones may lead to fistulous communications between the biliary passages and adjacent parts within the abdomen.*—Adhesions form between the gall-bladder and some adjacent viscus, and a communication is then established by ulceration advancing from the gall-bladder, or by gangrene.

a. *Fistulae into the Stomach* resulting from gall-stones have been rarely referred to, but Cruveilhier observed a case where a fistula between the gall-bladder and the stomach was found closed by a gall-stone.|| Oppolzer met with a case where the opening was

* Clin. Méd. ii. 536.

† Ser. xvi. No. 65.

‡ Med. Times and Gaz., 1859, ii. 372.

§ Brit. Med. Journ. May 30, 1868, p. 531.

|| Traité d'Anat. Path. ii. 541.

found close to the pylorus,* and two others are referred to by Frerichs.† I have already told you, that it is probably in consequence of this lesion that gall-stones are occasionally expelled by vomiting. Cruveilhier, indeed, is of opinion, that the very circumstance of a gall-stone being vomited is a positive proof of the existence of such a fistulous communication.

b. Fistulæ into the Duodenum are not very uncommon, and are almost invariably due to ulceration or gangrene of the biliary passages excited by gall-stones. The specimen which I hold in my hand is a good example of this fistula, and was obtained from the case I have already referred to, where death was due to ileus from the impaction of a large stone in the small intestine (Case XCII.). I have collected from different sources the notes of twenty-eight cases in which a similar fistula existed,‡ and in the majority of them the cause of death was the obstruction of the

* Zeits. der Gesellsch. d. Aerzte in Wien, Nov. 1860.

† Op. cit., ii. 525.

‡ Eight cases collected by Fauconneau-Dufresne, op. cit., p. 336; two observed by Cruveilhier, *Traité d'Anat. Path.* ii. 543; two by Oppolzer, *Zeitschr. der Gesellsch. der Aerzte in Wien*. Nov. 1860, p. 767; two by Frerichs, op. cit., vol. ii. Obs. 74 and 75; one by Duffin, *Lancet*, May 27, 1848; one by Blagden referred to by Duffin, but reference given is wrong; one by Peacock, *Trans. Path. Soc.* i. 255; one by J. W. Ogle, *ib.* v. 161; one by Pye Smith, *ib.* v. 163; one by Baly? *ib.* x. 185; one by Dr. Ezra Palmer, *Records Med. Soc. Boston, U. S.* vol. iii. p. 106; one in *Mus. Col. Surg. Path. Ser. No.* 1460; two? in *Barth Hosp. Mus. Ser.* xvi. No. 84 and ser. xix., No. 11; one in *St. Thos. Hosp. Mus.* No. 1412, and referred to in South's edition of *Chelius' Surgery*, i. 716; one in *Mus. King's College, Lond. Dig. Syst. Nos.* 57, 259, and 272; one in *Charing Cross Hospital Mus. G.* 3; one in *Mus. of Med. Soc. of Boston, U. S.* No. 565, *New Eng. Journ. of Med. and Surg.* 1825.

small intestine by a large biliary calculus which had escaped by the abnormal passage from the gall-bladder. In several, however, a large calculus was passed per anum after symptoms of obstruction, and, if the patient survive long the formation of the fistula, it is possible that this is overlooked after death, and that thus the lesion may be more common than is usually supposed. The opening in the biliary passages is almost always at the fundus of the gall-bladder, but occasionally, as in a case reported by Frerichs, it is in the common bile-duct;* that in the duodenum is in its third or lowest division. The size of the opening varies according to that of the stone which has been transmitted, and to the period which has elapsed between that occurrence and death. The symptoms attending the formation of these fistulæ are usually slight and obscure. The symptoms which have been noted consist mainly in vomiting, with colic and some tenderness in the region of the gall-bladder. Frerichs in one of his cases observed hæmatemesis and bloody stools. Jaundice is rarely present, for though the cystic duct may be obstructed, the common duct is usually free. It is only occasionally that the diagnosis will be assisted by a previous history of gall-stone colic with jaundice. After the formation of the fistula, there may be nothing to indicate its existence. Unless the gall-stone be large enough to cause obstruction of the bowels, the fistula may cause no symptoms of importance, and a biliary calculus may

* Op. cit., ii. 540.

be passed from the bowel with no trouble, which by the route of the bile-duct would have caused tremendous suffering; and thus we have an explanation of a statement of certain early writers, to the effect that small gall-stones cause more pain in their escape from the body than large ones. But if there be permanent obstruction of the common duct, and the cystic duct be free, the jaundice which has existed prior to the fistula will disappear, and the motions, unless there be obstruction of the bowels, will contain bile.

c. *Fistulæ into the Colon* from gall-stones are comparatively rare. Frerichs, Oppolzer, and all writers of authority agree in this statement. The exemption of the colon, as compared with the duodenum in this respect, is probably due to the former bowel being more movable. I have been able to find the records of only seven cases of fistulæ between the gall-bladder and colon, and in six of the seven there was cancer of the gall-bladder; * one of the six cases occurred in my own practice (Case XCVI.). The specimen of the seventh case is preserved in the Museum of St. Bartholomew's Hospital.† Here there were two fistulæ, one into the small intestine, the other into the colon: a large

* Two cases by Fauconneau-Dufresne, op. cit., p. 338; one by Durand-Fardel, Frerichs, op. cit., p. 480; one by Cruveilhier, *Traité d'Anat. Path.* ii. 543; one by Murchison, see Case XCVI. p. 543; one specimen in Mus. of Med. Soc. of Boston, U.S., No. 565. I have found only one instance of a cancerous fistula between the gall-bladder and duodenum, and there also the opening was closed by a calculus. Cruveilhier, op. cit., ii. 543.

† Ser. xvi. No. 84.

calculus had passed through the former into the ileum, and another, also large, into the colon and was found in the cœcum. In the body of a patient who died in this hospital some years ago, I found what was probably a similar condition in a more advanced stage, the gall-bladder communicated with the duodenum, the colon, and the external surface; there was no cancer, but the origin of the disease was probably a gall-stone (Case XCIV.).* But even when the fistula is cancerous it is probable that the ulcerative process leading to its formation is determined by gall-stones. In my own case there had been a previous history of gall-stone colic and jaundice, and in four of the remaining five cases gall-stones were found. In a specimen in the Museum at Boston, U. S., the patient, after symptoms of obstructed bowels, voided per anum, three months before death, a biliary calculus measuring $3\frac{3}{4}$ inches by 3 inches in circumference, and the signs of cancer did not show themselves till two months later. Of 11 cases of cancer of the gall-bladder collected by Frerichs, gall-stones were present in 9. The symptoms of cystico-colic fistula will be mainly those of the cancerous disease with which it seems to be usually associated. A simple cystico-colic fistula might be expected to cause less disturbance than a cystico-duodenal, as there would be less risk of a large calculus becoming impacted in the large bowel than in

* Dr. Bristowe has recorded a somewhat similar case where the common bile duct, in consequence of obstruction from gall-stones, communicated with the duodenum, the colon, and the portal vein. (Path. Trans. vol. ix. p. 285.)

the small. Its existence might be so little suspected during life, that possibly it has been sometimes overlooked after death, and that its occurrence may be more common than is supposed.

d. Fistulæ into the Urinary Passages.—There are at least two well-authenticated cases on record where biliary calculi have been voided with the urine during life, apparently owing to the formation of a fistula between the gall-bladder and the pelvis of the right kidney.* One of the patients passed nine small and four large calculi; the other voided 200 small calculi within a week; in both patients an operation was necessary to remove one of the calculi from the urethra. In both cases the calculi were analysed and found to consist of cholesterine and bile-pigment; and in one the analysis was made by Gmelin, who also found bile-pigment in the urine. Neither of the patients had ever jaundice; in both the symptoms were those of urinary rather than of hepatic disease; both recovered. In connection with this subject I may call your attention to the case of a patient who recently died of calculous pyelitis while under my care in the hospital, and whose urine always contained a large quantity of cholesterine and pus, although no communication existed between the urinary and biliary passages.†

e. Fistulæ into the Vagina.—The only instance with

* Fauconneau-Dufresne, op. cit., p. 341, and Gaz. Méd. de Paris Av. 18, 1840.

† This case is recorded in the Pathological Transactions, vol. xix.

which I am acquainted where a fistula formed between the biliary passages and the vagina is one quoted from Frank by Fauconneau-Dufresne, where an enlarged and inflamed gall-bladder contracted adhesions to a pregnant uterus, and burst into the vagina during parturition.*

f. Fistulæ into the Portal Vein.—It is a traditional statement that Realdus Columbus found three gall-stones in the portal vein of Ignatius Loyola, the founder of the order of the Jesuits, which had escaped from the gall-bladder by ulceration;† but although it may be doubted whether the concretions in that instance were not phlebolites,‡ there are several well-authenticated examples of fistulous communications between the biliary passages and the portal vein, with the presence in the latter of biliary concretions. Two such cases are referred to by Fauconneau-Dufresne§ and Frerichs, and a third is recorded by Dr. Bristowe in the Pathological Transactions (vol. ix. p. 285). In the last case also the common bile-duct opened into a cavity which communicated both with the duodenum and the colon. In these cases the common bile-duct is usually obstructed by a concretion, and the symptoms are those of portal obstruction—ascites or enlargement of the spleen, or both—supervening upon persistent jaundice.

g. Fistulæ into the Pleura.—One instance is on record

* Fauconneau-Dufresne, op. cit., p. 159.

† Frerichs, op. cit., ii. 526.

‡ Thudichum on Gall-stones, 1863, pp. 11, 268.

§ Op. cit., p. 340.

of a fistulous communication between the biliary passages and the pleura. This lesion was discovered by Dr. Cayley in the body of a patient who died in this hospital on March 2, 1866, while under the care of Dr. Thompson, and whose case is reported in the *Pathological Transactions* (vol. xvii. p. 161). All of the bile-ducts were enormously dilated, apparently in consequence of a gall-stone which had been impacted in the common duct, but which had escaped into the bowel before death. The common duct would allow the fore-finger to be introduced into it from the duodenum. The left pleural cavity contained more than a pint of bile mixed with pus, and in the left half of the diaphragm there was a perforation, large enough to admit a No. 4 catheter, which led into an irregular cavity between the left lobe of the liver and the under surface of the diaphragm, which in its turn communicated with a cystic dilatation of one of the bile-ducts in the liver. The case was also remarkable from the fact that there was no evidence of obstruction of the bile-duct until fifteen days before death, and that there were both the symptoms and the post-mortem appearances of acute or yellow atrophy of the liver.

12. *Gall-stones may be discharged from the biliary passages through fistulous openings in the abdominal parietes.*—The nine gall-stones which are in this bottle were discharged through an opening in the abdominal wall by a lady who is now under my care (Case XCIII.) ; and I find that in medical literature

and in pathological museums there are the records or the relics of at least 70 similar cases* which, with few exceptions, have occurred in females of middle or advanced age. These fistulous openings are formed in two ways. In some cases the ulcerative process which commences in the gall-bladder gradually eats its way through the adherent abdominal wall until it reaches the surface, while in others the gall-bladder becomes in the first place enormously enlarged from the accumulation of inflammatory products and opens externally, or, being mistaken for an abscess of the liver, is opened by the surgeon. The external opening is sometimes over the fundus of the gall-bladder, but very frequently it is at the umbilicus, to which it may

* Six cases observed or collected by J. L. Petit, *Mém. de l'Acad. Roy. de Chir.* 1743, tom. i. p. 255; eleven cases by Soemmering, *De concretis biliariis corporis humani*, 1795, p. 20; seventeen cases by Fauconneau-Dufresne, exclusive of three cases already reported by Petit. *Mém. de l'Acad. de Méd.* 1847, xiii. 320 and 167; three cases by Oppolzer, *Zeits. der Gesellsch. der Aerzte in Wien*, Nov. 1860, p. 747; three cases by Walter, *Frerichs on Dis. of Liver*, Eng. ed. ii. 525; three cases by Budd, *Dis. of Liver*, 3rd ed. p. 373; three cases in *Gazette des Hôpitaux*, 1846, Oct. 8, and 1847, p. 212; two by Cruveilhier, *Traité d'Anat. Path.* ii. 567, 570; two by Trousseau, *Clin. Méd.* ii. 534; two by Duplay and Fretin in *Archiv. Gén. de Méd.* 2me sér. i. 381, and 5me sér. iv. 86; one by Obre, *Path. Trans. of Lond.* i. 272; one by Simon, *ib.* v. 156; one by R. Robinson, *ib.* v. 158; one by Everet, *ib.* xviii. 120; one by Taylor, *ib.* xviii. 147; one by Heberden, *Comment.* 4th ed. p. 210; one by Santo-Nobili, *Schmidt's Jahrb.* lviii. 62, 1848; one by Schroeder, *Prag. Vierteljahrs.* xlv. Sup. p. 70, 1854; one by Callaway, *Lancet*, 1827-8, ii. 296; one by H. C. Stewart, *Lancet*, 1849, ii. 294; one by G. Robinson, *Med. Chir. Trans.* vol. xxxv. 1852, p. 471; one by Mackinder, *Brit. Med. Journ.* Dec. 26, 1857; one by Hinton, *ib.* Aug. 4, 1860; one by Cackle, *Med. Times and Gazette*, May 10, 1862; three specimens in *Mus. Roy. Coll. Surg. Cat. of the Calculi*, pp. 172, 176, 178; one in *Mus. of Med. Soc. of Boston*, U.S., No. 566.

be directed by the suspensory ligament of the liver; occasionally it is to the left of the middle line, in the inguinal region, or over the pubes as in a case observed some years ago at Paris, in which two biliary calculi were removed from above the clitoris, where they had been encysted in the subcutaneous tissue.* In rare cases there are two or more openings. The number of gall-stones discharged by the opening varies from one to upwards of 600. When there is only one, it may be as large as a hen's egg. Fauconneau-Dufresne refers to one which measured 3·15 inches in length by 1·1 inch in width. Calculi may be discharged almost as soon as the opening is formed, or not for years afterwards. The fistula has been known to keep discharging for many months after all the calculi have come away, but usually it soon closes after this has occurred, if there be no bile in the discharge. The fluid which drains away from the fistula may be pure bile, the daily quantity of which has been found to vary from 8 oz. to 2 pints.† Far more commonly it is pure pus, or a glairy mucus with now and then a little blood. In most cases the cystic duct is obstructed and then no bile can escape by the fistula; in a few cases the common duct only is obstructed, and then the jaundice caused by this obstruction disappears in a great measure on the formation of the fistula, although no bile is present

* Gaz. des. Hôp. Oct. 8, 1846.

† Fauconneau-Dufresne, op. cit., p. 325; and G. Robinson, Med. Chir. Trans. xxxv. 471.

in the stools ; in rare cases the gall-ducts are patent and bile passes off both by the fistula and the bowels. After the discharge of a gall-stone, the external opening rapidly contracts and is drawn inwards, while the fistulous passage connecting it with the gall-bladder may be several inches in length and may also be extremely tortuous and surrounded with callous induration. Not unfrequently gall-stones become lodged in a cul de sac of the fistula, or may completely obstruct it and lead to the accumulation of matter behind and the formation of fresh abscesses. The adhesions between the gall-bladder and the abdominal parietes may be very extensive, but are often very limited. These fistulous openings are chiefly serious from the inconvenience which they occasion. A large proportion of the patients survive their formation for years and enjoy good health, and in many the fistulæ completely heal. This last event may be predicted with most confidence when there is only one large calculus, when the external opening is directly over the gall-bladder, when the discharge from it contains no bile, and when there is no jaundice. There is little hope of the fistula permanently closing while calculi still remain in the gall-bladder, or if the cystic duct be patent while the common duct is closed. In cases where the quantity of bile lost is great, which are happily rare, the patient may become rapidly reduced in flesh and strength and die of marasmus. When the calculi are many in number, and the fistula is long, tortuous, and surrounded by

callous tissue, the opening though small may continue for years, showing every now and then a tendency to cicatrize, but again enlarging on the escape of a fresh calculus, the passage of which often entails much suffering, although in the intervals the patient enjoys good health.

From this lengthened list of the evils of which gall-stones may be the source, you will learn to avoid the common error of regarding them as a harmless, though perhaps painful malady, and you will see the necessity in all cases of being guarded in your prognosis.

Treatment of the consequences of Gall-stones.

1. When the symptoms indicate the presence of gall-stones in the gall-bladder, the treatment must consist in those constitutional measures which I have already told you are useful for gall-stones in general (p. 352), and in remedies for correcting any symptoms of indigestion. At the same time the patient should be cautioned as to the risk of a gall-stone being projected into the bile-ducts by sudden or severe muscular exertion, or by driving over a rough road, especially after meals.

2. The measures to be adopted when gall-stones are in the ducts have already been described to you in detail (p. 349). When there is reason to believe that the calculus has entered the bowel, it is well to administer some laxative such as castor

oil, with the object of expediting its passage to the anus.

3. When symptoms of inflamed gall-bladder—fever with pain, tenderness, and often a distinct tumour—supervene upon those of gall-stones in the ducts, the treatment may be summed up in the three words which I mention in the order of their importance, viz:—rest, opium, and leeches. Rest is all important to promote the formation of adhesions and prevent the extension of the inflammation to the general cavity of the peritoneum. Any sudden movement may produce a rupture in the inflamed and softened gall-bladder, and fatal peritonitis in consequence. Opium may be given in large and repeated doses, and the pain will often be materially relieved by the application of a few leeches below the right ribs. The expediency of puncturing the gall-bladder, when it is very large, will be considered presently.

4. When symptoms of gall-stones in the gall-bladder or ducts, or of inflammation of the gall-bladder, are followed by those of ileus, although death is too often the result, recovery after the expulsion of a large calculus per anum has been sufficiently frequent to justify us in sparing no effort for the attainment of this desirable end. Warm baths and fomentations, opium in full and repeated doses, and copious enemata of warm water and oil are the measures most to be relied on. Gentle pressure and manipulation of the abdomen have been thought in some instances to have displaced the stone. Sir Thomas Watson relates how

a lady suffering from ileus experienced a sensation as if this had occurred during her examination by three medical men in succession. While they were still consulting she had a liquid motion precisely resembling what she had last vomited, and next day she voided a gall-stone as big as a walnut.* Lastly, in all cases where there is reason to believe that intestinal obstruction is due to biliary concretions, it will be well to examine the rectum. Now and then the obstruction is situated immediately above the sphincter and can be removed by the finger or scoop.

5. Internal biliary fistulæ are beyond the reach of medical or surgical art, but those into the intestines which are most common are scarcely dangerous except from their sometimes permitting the escape of a calculus large enough to obstruct the bowels.

6. When external biliary fistulæ discharge one large rounded calculus without facets, and the outer opening is over the fundus of the gall-bladder, they usually heal speedily without any interference. But when the gall-bladder contains many small calculi, and still more when the fistula is long, narrow, circuitous, and surrounded by callous tissue, the opening may remain for years or may never close, and every now and then the fistula is apt to become blocked up by a calculus, the passage of which causes much pain and leads to accumulation of matter behind it. Under these circumstances, the question will arise as to the expediency of dilating or slitting up the fistula

* Lect. on Pract. of Physic, ii. 465.

to facilitate the extraction of the calculi. This has been done in many cases with a successful result; but on the other hand there are several instances on record, where even slight interference, such as the introduction of a dressing forceps, has brought on fatal peritonitis. There is no general rule applicable to all of these cases, but the question of operating must be decided by the peculiarities of each case. If, on probing, a calculus can be felt near the outer opening and is long delayed there, it ought to be extracted; but if no calculus can be felt, and still more if the fistula take an inward direction towards the peritoneum, the risk of interference ought to counterbalance the inconvenience of the fistulæ, and it is better to wait. When pure bile drains away in large quantity from the opening, and none enters the bowel, there is little chance of the fistula ever closing, and it is not desirable that it should; but if the common bile-duct be patent, and the patient suffer from the exhaustion consequent on the external drain of bile, the question of closing the fistula may fairly be entertained.

In illustration of some of these evil consequences of gall-stones, I may now bring under your notice the following cases:—

CASE XCI.—*Gall-Stones in a sacculus of the Common Bile-Duct and in the Gall-Bladder—Ulceration and Perforation of the Gall-Bladder—Fatal Peritonitis.*

Mrs. C——, aged 55, had consulted me repeatedly during three years, for noises in the head, and other distressing nervous

symptoms, which first appeared after a period of great mental anxiety, and which could only be accounted for by a weak state of the circulation, with probably a fatty heart. She had in consequence taken but little exercise, and spent a great part of her time in bed. About the end of May, 1867, I was called to see her, and found that she had decided jaundice of the skin and conjunctivæ, and that the urine contained a considerable amount of bile-pigment. The liver also was slightly enlarged. For two or three days she had been suffering from paroxysms of severe pain in the right hypochondrium with vomiting. A feeling of soreness remained in the intervals of the paroxysms, and there was slight tenderness below the right ribs. The pulse, however, was only 72, and the skin was cool. Under the use of warm fomentations, repeated doses of morphia and laxatives, the acute symptoms subsided in a few days; by the end of a fortnight, the jaundice had disappeared, and the patient was able to go out.

On June 24 she had a return of the severe pain in the abdomen, and when I saw her on the 26th she was again jaundiced, and her symptoms differed from those in the previous attack in that her pulse was 96; the skin felt slightly hot, and there was rather more tenderness below the right ribs, with a tendency to hiccup. Still the pain was for the most part paroxysmal, and relief was again obtained from morphia. For two days she seemed to improve, but on the night of the 28th she became rather suddenly worse, and at my visit on the following day she had all the symptoms of acute peritonitis. The pulse was 136, small and feeble; the respirations were short, quick, and thoracic; and there was constant vomiting and hiccup. The abdomen was greatly distended and tympanitic, and there was now acute pain and tenderness, chiefly in the left side—constant and aggravated by the slightest movement. From this time the patient continued to sink until death occurred on the night of July 1.

At the *post-mortem* examination, two openings were found in the fundus of the gall-bladder, both with ragged edges, and one large enough to admit the finger. Through these openings bile had escaped in large quantity into the peritoneum. Firm adhesions of the great omentum to the abdominal wall had directed the bile entirely to the left side of the abdomen, where there were signs of recent peritonitis—intense vascular injection, and lymph coloured with bile. The mucous surface of the gall-bladder surrounding the openings was extensively ulcerated, apparently from the pressure of a gall-stone, the size of a cherry, which was in imme-

diate apposition, and had not escaped into the peritoneum. The common bile-duct communicated with a pouch, as large as a hen's egg, containing bile and upwards of a dozen polyhedral gall-stones, each about the size of half a cherry; but no concretion was found obstructing the duct between the pouch and the duodenum. There were no abscesses in the liver. The heart, kidneys, and liver were in a state of fatty degeneration.

CASE XCII.—*Fistulous Opening between the Gall-Bladder and the Duodenum—Fatal Obstruction of the Small Intestine by a large Biliary Calculus.**

A. Mc D—, aged 46, was admitted into the Middlesex Hospital under the care of Dr. Stewart, on January 29, 1856. Her general health had been good, but for many years past she had been of a costive habit, the bowels being seldom moved without taking aperient pills. She had been subject to bilious attacks with vomiting of green bitter matter, and to loss of appetite, with excessive flatulence which increased after meals, but she never had jaundice.

Twelve days before admission she took two 'antibilious pills,' which acted freely on the following day. Vomiting took place at the same time and continued with little intermission ever afterwards. The motions and vomited matters were of a green colour. Sleep was much disturbed. Two days after this she was suddenly seized with a severe, sharp pain in the right iliac region, where the tenderness on pressure was still acute. This pain continued with remissions until the time of admission. She had no motion for ten days from that time, *i.e.* from the 19th to the 29th of January.

On admission, an injection consisting of a pint of gruel and some castor oil was administered. It passed up entirely, but caused great pain, and was soon discharged with several large hard scybalous masses. The abdomen then became flaccid; it was slightly dull to the right of the umbilicus, but elsewhere perfectly resonant. Her countenance was somewhat pinched and anxious, and the cheeks flushed. Tongue dry, and covered with a thick yellow fur on the dorsum, moist and clean at the edges; thirst

* This case was reported by Dr. Vanderbyl to the Pathological Society (vol. viii. p. 231).

very urgent. Pulse 80, very small. Slight, sonorous rhonchi on the right side of the chest; breathing healthy on the left side. Heart-sounds normal. A warm bath was ordered, to be followed by poppy-head fomentations to the abdomen and an opiate pill.

January 30.—Passed a tolerable night, and notwithstanding a constant sensation of nausea, has not vomited since admission. Two pints of injection passed up without difficulty or pain, but after being retained half an hour returned without a trace of fæculent matter. The abdomen is now much more tense and tympanitic, and is still painful on pressure.

10 P.M.—Countenance less anxious; thirst very great; nausea continues, but she has not vomited until just now, when she suddenly raised herself on her elbow and vomited more than half a pint of dark brown and very offensive stercoraceous fluid. The retching continued for several minutes until she had brought up about three pints of this fetid fluid. Pulse 78.

January 31.—There was no recurrence of the vomiting till 11 A.M. when she vomited about a pint of fluid having the same colour, but without the fæculent odour of that brought up yesterday.

The abdomen became more and more distended; the pain increased; no motion passed; there was frequent retching; exhaustion supervened, and she died rather suddenly this evening.

Autopsy.—On opening the abdomen the intestines appeared very much distended, and were in some places adherent to each other by recent lymph; the great omentum was puckered up and adherent to the intestines. On separating the intestines, a solid body about the size and shape of a cork was found to block up the small intestine, about the middle of the ileum, which it fitted like a plug. At the point of obstruction the gut was bent upon itself, the adjacent peritoneal surfaces being slightly adherent by recent lymph. The intestine seemed to become suddenly smaller immediately below the obstruction, but above this point it was greatly distended and filled with dark, greenish, fæculent matter, in which were found ten small angular, biliary calculi, of about half the size of hazel-nuts. The dilated portion of the intestine was dark, and when laid open the mucous membrane was found much congested, and in some parts coated with adherent false membrane; a number of small roundish ulcers were scattered over the surface. The obstructing body was discovered to be a large biliary calculus. It was perfectly cylindrical, measuring nearly

four inches in circumference, and one inch and a quarter in diameter. Its external surface was uniformly nodulated, the extremities being rather smooth. When divided transversely it exhibited a crystalline appearance.

Below the obstruction the intestine was much contracted, and pale throughout, containing only a little thickish mucous secretion. The colon was almost empty. The stomach contained some greenish fæculent matter, and one small angular biliary calculus. The gall-bladder was firmly adherent to the duodenum at the point where it turns down to become perpendicular, and a well-defined communication existed between these two parts, large enough to admit a finger easily. The gall-bladder was contracted and converted into a small fibrous pouch, but there could be no doubt that the calculi passed through this perforation, although the opening was now much smaller than the calculus causing the obstruction. The common bile-duct was patent but not dilated; the cystic duct was closed. The liver weighed 58 ounces, and appeared tolerably healthy, though rather dark. The heart, lungs, and kidneys were not diseased.

CASE XCIII.—*Closure of the Cystic Duct—Abscess of the Gall-Bladder—Discharge of Gall-Stones through a fistulous opening in the Abdominal Parietes.*

On June 18, 1867, the Countess —, about 54 years of age, consulted me on account of a fistulous opening in the abdominal parietes, and gave me the following history of her illness, which was subsequently supplemented by Mr. Bickersteth of Liverpool, who had previously attended her. In the previous autumn she had suffered from an attack of biliary colic with jaundice and vomiting, which passed off after two or three weeks. Towards the end of the year she was attacked with persistent pain in the region of the liver, which gradually increased, and was attended by considerable fever and other symptoms of constitutional disturbance, but not by jaundice. Very soon an enlargement was noticed in the right hypochondrium, and early in February there were all the signs of a deep-seated abscess below the right ribs, while the patient's general condition was such as to excite considerable alarm. The abscess was opened by Mr. Bickersteth with *potassa fusa*, the integuments having been previously divided.

Nearly a pint of 'tolerably healthy pus not mixed with bile escaped after a few days.' All the hepatic symptoms ceased and she slowly recovered strength. Some weeks afterwards, another small superficial abscess was opened at the umbilicus. The second opening soon closed, but the first continued to discharge small quantities of pus and of a glairy fluid, and sometimes a little blood.

This opening was situated about two inches above and to the right of the umbilicus, and at about an equal distance from the natural situation of the fundus of the gall-bladder. It had a diameter of about two lines, was slightly depressed below the surface, and was surrounded on all sides for about two inches by considerable induration of the abdominal parietes, which, in an upward direction, amounted almost to a stony hardness. On introducing a probe, the fistula seemed to take an upward direction, but as the instrument caused pain and slight bleeding it could not be carried further in than half an inch. The patient complained of dragging pains round the opening and in the right hypochondrium, and of occasional attacks of nausea and headache, but there were no other indications of constitutional disturbance. From the symptoms, I expressed the opinion at my first visit that the abscess had not been in the liver, but in the gall-bladder, and I ventured to predict that sooner or later this would be proved by the discharge of gall-stones through the fistulous opening. Poultices were kept constantly applied over the opening, and quinine with nitric acid, and occasionally purgatives, were ordered to be taken internally.

On July 28, the first gall-stone came away through the opening. It was about the size of a pea and presented several facets. Its passage through the fistula was attended for several days by considerable pain, and its escape was followed by the discharge of a good deal of thick yellow pus.

On August 15, three more concretions came away, one somewhat larger than the first, the two others very small. There was again for some hours a good deal of pain, and after the exit of the calculi a discharge of thick matter with a small quantity of blood.

In September and October, two or three more concretions escaped, and on December 19, after two days and nights of intense pain a polygonal calculus found its way out, which was fully half an inch in diameter. Another smaller concretion (the ninth) escaped in January, 1868.

Since January till the present time (May 7) no calculi have appeared. The induration surrounding the opening has greatly diminished, but there is still a mass of stony hardness, about the size of a walnut, immediately above it. The opening is not more than a line in diameter and is retracted to the bottom of a deep depression with puckering and induration of the surrounding skin. This change is mainly due to the greater thickness of the abdominal wall from the deposit of fat. Several times since the escape of the last stone and in the intervals of the escape of the previous ones, the opening has shown a tendency to close and caustic has been applied. On one occasion an attempt was made to dilate the fistula by a tent of sea-tangle, but the removal of the swollen tent from the tortuous tract caused so much pain that the procedure was abandoned. The patient has gained flesh and strength, looks extremely well, has a good appetite and digestion, and only suffers from the inconvenience of the discharge from the fistulous opening.

[Early in June another calculus escaped, and shortly afterwards the patient suffered for a few days from febrile disturbance, apparently due to a temporary retention of the discharge from the fistula.]

The following case occurred while I was pathologist to the hospital, and was recorded by me in the 'Pathological Transactions' (vol xii. p. 85). The sequence of events was probably as follows:—

1. Ulceration of the interior of the gall-bladder from the presence of a gall-stone, perforation of its coats, and the formation of fistulous communications with the duodenum and colon, by which the calculus escaped into the bowel, as in the specimen in St. Bartholomew's Museum already referred to (p. 516).

2. Rupture by straining of some of the adhesions between the bowels, and the formation of a circumscribed fæcal abscess which opened externally.

3. Blood-poisoning, pyæmic abscesses, lobular pneumonia, and pericarditis.

CASE XCIV.—*Fistula in the Abdominal Parietes opening into a circumscribed cavity which communicated with the Colon and Duodenum, and indirectly with the Gall-bladder.*

B.Z—, aged 38, was admitted into the Middlesex Hospital under Dr. Goodfellow on September 25, 1860, and died on November 14. Married twice; eight children and six miscarriages.

About a year before death, without any apparent cause, she was suddenly seized with sickness, vomiting, and great prostration, which symptoms were followed by general fever and tenderness over the abdomen. The bowels were regular. After a few weeks, she recovered and remained in her ordinary health up to September 14, 1860.

On September 13 she fancied that she strained the abdominal muscles by carrying some heavy pails of water upstairs. On the following morning she awoke with slight pain in the abdomen, which was greatly increased after breakfast, and was then accompanied by sickness and vomiting of a green bitter fluid. She said the pain was just as if her abdomen had been tied round with a rope. The sickness abated after three days, when the bowels had been freely opened by medicine, but the pain continued and the patient became very weak. On her admission into hospital on the 25th, there was great tenderness of the abdomen, which was most intense at the umbilicus. Immediately to the left of the umbilicus, was a superficial circular swelling with a firm dense rim and doughy in the centre. Motions of bowels normal. Pulse 144. Great prostration.

The next day (26th) a little yellow pus of a stercoraceous odour, but exhibiting nothing except pus-cells under the microscope, could be squeezed through the umbilicus. After this date the opening continued to discharge large quantities of fetid pus. From September 30 to October 17, the pus was mixed with faecal matter. On November 9 the opening ceased to discharge even pus.

About three weeks before death, abscesses began to form in various parts of the body, over the right parotid, in the soft parts of the right hip, &c., and the patient suffered from great dyspnoea and expectorated purulent sputa. It was impossible to examine the chest, as the slightest movement or manipulation caused great

pain. The prostration gradually increased, and death took place on November 14.

Autopsy.—Body greatly emaciated. An abscess containing seven or eight ounces of pus in the right hip. At the umbilicus there was a fistulous opening large enough to admit a goose-quill. This opened into a sloughy cavity the size of a small orange, which communicated with the transverse colon and the duodenum, and indirectly with the gall-bladder. The opening into the colon was large enough to admit the finger; the colon at this place was much constricted, and its lining membrane injected and slightly ulcerated. Immediately to the right of this opening the gall-bladder was firmly adherent to the colon. The gall-bladder was small and contained about two drachms of whey-like fluid, without any tint of bile. The cystic duct was obliterated, but there were no gall-stones. Between the fundus of the gall-bladder and the colon was a fistulous communication running somewhat obliquely, and just large enough to admit a No. 1 catheter. The inner surface of the gall-bladder around this opening was marked by an extensive radiated cicatrix. The fistula between the sloughy cavity and the duodenum was large enough to admit a crow-quill and opened into the duodenum immediately beyond the pylorus. The abdominal parietes around the sloughy cavity were inseparably adherent to the viscera, and there was no fluid in the peritoneum. About one pint of clear serous fluid in the left pleural cavity. A few old adhesions over apex of left lung, which for the most part was normal, but the lower lobe contained several nodules of lobular pneumonia, the largest about the size of a walnut, grey, granular, bulging above surface on section and very friable. Two or three pints of turbid serous fluid containing numerous flakes of lymph in right pleural cavity, and lung glued to walls of chest anteriorly and at apex by recent lymph. Lower lobe of right lung collapsed, non-crepitant, sinking in water, smooth on section, and very tenacious.

Fully eight fluid ounces of a gelatinous, yellowish, opaque, puriform substance in pericardium, which could be scooped out in one semi-solid mass. This substance, on microscopic examination, was found to consist of fine fibrillated material with numerous lymph or pyoid corpuscles, but no true pus-cells with characteristic nuclei. Pericardium inseparably adherent to left ventricle over a space measuring an inch and a half in diameter. Outer surface of heart covered with membranous patches of lymph, many of which were firmly adherent.

C. ENLARGEMENTS OF THE GALL-BLADDER.

In the diagnosis of diseases of the liver it is important to keep in view the various causes of enlargement of the gall-bladder. Before closing this lecture I shall therefore say a few words on the distinctive characters of these enlargements, which, for clinical purposes, may be said to be due to five causes: viz.: I. Accumulation of Bile; II. Suppuration; III. Dropsy; IV. Gall-Stones; V. Cancer.

I. *Enlargement of Gall-Bladder from Accumulation of Bile.*

Enlargement of the gall-bladder from accumulation of bile, as I have already told you (pp. 142, 322), is one of the first consequences of obstruction of the common duct, and it is then distinguished by the following characters:—

1. Jaundice, which gradually becomes intense.
2. Absence of bile from the motions.
3. General enlargement and tenderness of the liver (see p. 142).
4. An elastic or fluctuating, pear-shaped, somewhat tender tumour, projecting from the edge of the liver in the situation of the gall-bladder. Dr. Bright has recorded a case where the gall-bladder in this condition formed a fluctuating tumour extending almost to the crest of the ilium,* but its dimensions are not often so great. This tumour may suddenly subside

* Abdom. Tumours, Syd. Soc. Ed. p. 271.

with the discharge of a large quantity of bile in the motions and the disappearance of the jaundice; but even when the obstruction of the bile-duct is permanent, the bile in the gall-bladder is often gradually absorbed and after a time a condition of atrophy may be substituted for that of dilatation. Sometimes the distended gall-bladder, when its coats are softened by inflammatory action or fatty degeneration, will rupture and cause fatal peritonitis, as in Case XCI.

Rare cases have been recorded where a gall-stone in the neck of the gall-bladder, or in the cystic duct, has acted like a plug-valve, permitting bile to enter the gall-bladder, but preventing its exit, and where bile has in consequence accumulated in the gall-bladder. In such cases there need be no jaundice nor clay-coloured motions, but the occurrence is so rare as not often to embarrass the diagnosis.

In former lectures I have brought under your notice several instances of enlargement of the liver from accumulation of bile (Case XXXI. p. 145, Case LXI. p. 365, Case LXII. p. 367, and Case LXIV. p. 371).

II. *Enlargement of the Gall-Bladder from Suppuration.*

The gall-bladder occasionally becomes distended with pus which may be mixed with bile, or may be indistinguishable from that of an ordinary abscess as in Case XCIII. Inflammation of the gall-bladder may follow its over-distension with bile from obstruction of the common duct; but in most cases of suppuration it is the cystic duct only that is obstructed, and

the inflammation is limited to the gall-bladder, and is due to the irritation of gall-stones, or to some other cause. It is then characterized by:—

1. A tumour corresponding in situation and shape to that caused by distension with bile, but more painful and tender, and accompanied by more febrile disturbance, and sometimes by rigors and night-sweats. There are in fact all the characters of hepatic abscess, from which even its shape and situation may not suffice to distinguish it.

2. There is no jaundice.

3. The motions contain bile.

4. There is no general enlargement or tenderness of the liver.

5. Occasionally as in Case XCIII. there is a previous history of biliary colic.

6. It is only the tropical abscess of the liver (see p. 165), which is simulated by suppuration of the gall-bladder, and accordingly the diagnosis may be assisted by the circumstance of a tumour answering to the description now given occurring in a temperate climate.

III. *Enlargement of the Gall-Bladder from Dropsy* (*Hydrops Cystidis Felleæ*).

When the gall-bladder is distended with pus it may open externally and form a biliary fistula, or it may burst into the peritoneum or into the bowel. But occasionally a thin flaky liquid appears to be substituted for the pus; or sometimes, from the inflammatory process being slight and chronic, the fluid has these

characters from the first. This is what is meant by dropsy of the gall-bladder. It is not a *dropsy* in the strict sense of the word, but a chronic inflammation. Enlargement of the gall-bladder from this cause has all the characters of enlargement from suppuration, except that it is scarcely, if at all, painful, and that it is not necessarily accompanied by febrile disturbance. This consequently is the form of enlargement of the gall-bladder, which is most readily mistaken for a pendulous hydatid (see p. 60), from which it is to be distinguished mainly by its situation, and by the fact of its development being often, though not necessarily, preceded by a history of biliary colic.

IV. *Enlargement of the Gall-bladder from accumulation of Gall-stones.*

Gall-stones sometimes accumulate in the gall-bladder in such quantity as to form a distinct tumour (see p. 501). We have now in Cambridge ward a patient with a tumour probably due to this cause (Case LXIV. p. 371). This form of enlargement may be recognized by these characters:—

1. It is hard and sometimes nodulated.
2. It is usually movable.
3. Although often a centre of uneasy sensations (see p. 500), it is painless on pressure.
4. Occasionally a crackling sensation is experienced on manipulating the tumour, or the patient complains of a sensation of a weight rolling from side to side when he turns in bed (see p. 500).

5. There is in most cases either jaundice, or a previous history of biliary colic.

6. Its size does not vary, or its growth is slow and imperceptible.

7. The usual indications of cancer are absent.

These characters may be modified when the gall-stones excite ulceration of the mucous membrane or local peritonitis. The tumour may then become painful and adherent and may increase in size.

V. *Enlargement of the Gall-bladder from Cancerous Deposit in its walls.*

Cancer of the gall-bladder is sometimes secondary to cancer of the liver or pancreas (Case LXIII. p. 369) or of some more distant organ (Case XCV.); at other times the disease seems to commence in the gall-bladder, and the peritoneum or liver is affected secondarily (Case XCVI.). It is remarkable that in most of these cases the gall-bladder contains calculi (see p. 517). Enlargement of the gall-bladder from cancer has the following characters.

1. There is a hard sometimes nodulated tumour, about the size of an orange, more or less, in the region of the gall-bladder. Occasionally the tumour feels soft in the centre from softening of the cancerous matter, or from the cancer being chiefly at the neck while the fundus contains fluid (Case XCV.).

2. It is adherent and immovable.

3. It is very tender on pressure, and is usually the seat of severe lancinating pains.

4. Its growth may be rapid.

5. Jaundice and vomiting are common symptoms owing to the extension of the cancer to the common bile-duct, or to the pressure of the tumour on the pylorus.

6. Fistulous communications with the digestive canal, and particularly with the colon, are not uncommon, and consequently the passage of a large gall-stone, with or without hæmorrhage per anum, concurring with a tumour like that now described, would corroborate rather than refute the diagnosis of cancer (see p. 517).

7. There is rapid emaciation with the usual phenomena of the cancerous cachexia.

Treatment of Enlargements of the Gall-Bladder.

1. The treatment of over-distension of the gall-bladder with bile has been already considered under that of jaundice from obstruction of the bile-duct. (See p. 356.)

2. In the course of this lecture I have told you what measures you must have recourse to in inflammation of the gall-bladder. It now only remains for me to add :—

a. That in all such cases the patient must be cautioned against the risk of a severe muscular strain or a slight blow. Cases have been recorded where from such causes, the gall-bladder has been ruptured and fatal peritonitis has been the result.*

* There is a preparation showing this in the Museum of St. Bartholomew's Hospital, ser. xix. No. 14.

b. That now and then it will be necessary to puncture the gall-bladder and evacuate its contents (see Case XCIII.), but that this ought never to be done except when the tumour is growing so rapidly that there is imminent danger of its bursting, or the constitution is being worn out by hectic fever. From what has been stated it is also obvious that the operation is rarely advisable when there is jaundice with absence of bile from the motions. If there be no adhesions over the tumour it will be necessary to produce them by means of caustic potash.

3. Accumulations of gall-stones in the gall-bladder must be treated in the manner already described (see p. 352) ; and lastly,

4. In cancerous enlargement all that can be done is to relieve distressing symptoms and promote euthanasia.

In conclusion, I may bring under your notice the two following cases of cancer of the gall-bladder, which came under my observation a few years ago, and were reported by me in the eighth volume of the *Pathological Transactions*. In one case the cancer of the gall-bladder was secondary to cancer of the rectum and liver ; in the other, the cancer of the liver appeared secondary to that in the gall-bladder.

CASE XCV.—*Cancer of the Rectum—Secondary Cancer of the Liver involving the Gall-Bladder and obliterating the Cystic Duct—Enlargement of Gall-Bladder.*

A. B——, a female aged 53, was admitted into St. Mary's Hospital on August 29, 1856. She had been suffering from pains in the loins and abdomen for two months, and on admission she had also slight jaundice and constipation. The liver was not enlarged, but a tumour, of the size of a small orange, projected from the lower border in the site of the gall-bladder. These symptoms increased, and a week after there was superadded uncontrollable vomiting. All treatment proved unavailing; the jaundice became more marked, the constipation more confirmed, and the patient grew gradually weaker till her death on September 28.

On *post-mortem* examination, small nodules of cancer were found scattered over the peritoneal surface of the intestines. There was a stricture of the rectum from similar deposit commencing one inch and a half from the anus and extending upwards for three inches. The only portion of the liver affected was the lobus quadratus. In this there was a cancerous deposit, the size of a small orange, which involved, and had obliterated, the cystic duct. The coats of the gall-bladder for one-fourth of their extent from the duct were thickened by deposit. The anterior three-fourths appeared free from disease. The whole gall-bladder was of a pale colour, and much distended, so as to project two inches in front of the anterior margin of the liver. On opening it, it was found to contain a milky flaky fluid exhibiting under the microscope numerous epithelial scales and two gall-stones about the size of marbles. There was no trace of ulceration in any part of its lining membrane, and no adhesion between its outer surface and any of the viscera. Bile was found in the duodenum. The mucous membrane of the stomach, the spleen, the kidneys, and the lungs were free from disease.

CASE XCVI.—*Destruction by Cancerous Ulceration of the Gall-Bladder and communication of the resulting cavity with the Transverse Colon—Cancer of the Liver.*

S. P——, aged 56, a coach-painter, came under my care on August 14, 1856. He stated that, when a young man, he had an

attack of jaundice preceded by severe cramps in the stomach. Fourteen years before I saw him he had suffered from rheumatic fever, followed by palpitations and other symptoms of cardiac disease. His father had lived to a great age, and his mother died at 86 of cancer of the uterus. He was his mother's last child, and was born when she was nearly 50.

Three months before he applied to me for relief he began to suffer for the first time from a pain in the region of the liver, but he continued regularly at his work till the middle of July. About this time he was seized with severe abdominal pains, vomiting, and purging. He continued at work, although irregularly, for a fortnight longer, but on the 1st of August he was obliged to take to bed. On August 7, he became jaundiced. The following notes were taken when he was seen by me on August 14. 'Is very much emaciated, and conjunctivæ are of a deep yellow colour, and the countenance has an anxious cachectic aspect expressive of inward pain. The tongue has a yellowish fur; vomiting still continues, almost everything he swallows being immediately rejected, and sometimes apparently before it reaches the stomach. Bowels relaxed. Two or three days ago, his wife states that he passed by stool a quantity of black matter like blood. He complains of severe pain in the hepatic region, coming on at intervals, and of a sharp shooting character. Hepatic dulness in the right mammary line extends four inches below the margin of the ribs, and a tumour can be felt in the region of the gall-bladder, two or three inches in diameter, immovable, apparently connected with the liver, and very painful on pressure. The patient does not sleep on account of the pain; pulse, 100. A diastolic blowing murmur is heard over the middle of the sternum.'

He was ordered milk diet, wine, opiates, and various remedies to check the vomiting, including naphtha and dilute hydrocyanic acid. Nothing, however, proved of any avail; he gradually sank, and died on August 19. Previous to death his stools had assumed a perfectly natural colour and consistence.

Autopsy.—There were some opaque patches on the surface of the heart and several small vegetations on both the mitral and aortic valves. The lungs were healthy.

There was no effusion into the peritoneal cavity, and the spleen and kidneys were normal. Scattered throughout the substance, and over the surface of the liver, were a number of small white masses of morbid deposit, varying in size from that of a pea to

that of a small orange. Those on the surface were umbilicated in the centre, and from all of them exuded on pressure a milky juice containing a multitude of 'cancer-cells' and free nuclei. These cells varied in size from $\frac{1}{320}$ to $\frac{1}{1500}$ of an inch, and in form were rounded, elliptical, fusiform, pear-shaped, &c., while their nuclei were large and well defined. Among them were a few 'mother-cells.' The transverse colon was firmly adherent to the anterior margin of the liver, at a part corresponding to the situation of the gall-bladder; and on slitting up the bowel, its interior was found to communicate by an opening as large as a half-penny piece with a cavity hollowed out in the substance of the liver, measuring two inches and a half from before backwards and one inch and a half from side to side. The walls of this cavity presented an irregular sloughy aspect, being composed of disintegrated hepatic and cancerous tissue, and its interior was filled with a dark-brown pultaceous fluid containing a piece of potato-skin and other *débris* of the food. This cavity corresponded exactly in position to the site of the gall-bladder, no trace of the walls of which could be seen. The remains of the obliterated cystic duct and artery were made out imbedded in a mass of cancerous deposit the size of a Spanish chestnut, which also compressed, but did not obliterate, the common hepatic duct. The margins of the opening in the transverse colon and the whole circumference of the corresponding portion of the bowel were thickened by deposit which narrowed the calibre of the gut, so as to produce a stricture barely admitting the point of the finger. The bowel above this stricture was much dilated, and exhibited on its mucous surface a number of superficial circular ulcers, the largest being about the size of a silver penny-piece. The continuation of the gut beyond the stricture was contracted. There was no disease of any other portion of the intestines or of the stomach, but the pylorus was compressed by the cancerous deposits in the liver.

APPENDIX.

SINCE Lecture IX. has passed through the press, important changes have taken place in the symptoms of William M——. (Case LXIV., p. 374), which deserve to be noted.

July 4, 1868.—During the last fortnight the patient has been losing flesh and strength, and he is now so weak as to move in bed with difficulty. He has lost all appetite for food, and at intervals of two or three days he has vomited a quantity of brown yeasty matter containing sarcina. The motions are no longer clay-coloured, but sometimes they have been almost black, and at other times reddish-brown, apparently from the admixture of altered blood. The hepatic dulness in the right mammary line does not exceed $2\frac{1}{2}$ inches; but large, elevated, flattened nodules can be felt over the surface of the liver in the epigastrium, and are even visible through the attenuated abdominal parietes. One on the right side of the middle line is about the size of a florin; another on the left is a good deal larger: they are not tender. The tumour below the right lobe of the liver has increased considerably in size, but is still hard, movable, and painless. The urine on evaporation yields globular crystalline masses of tyrosine; it also now contains a little albumen and crystals of oxalate of lime.

INDEX.

ABD

ABDOMINAL parietes, abnormal states of, in relation to diseases of liver, 20

— — abscess in, 21

— — fistulous openings into gall-bladder, 520, 531, 534

— — fistulous openings into hydatid of liver, 65, 90, 94

Abscess between diaphragm and liver, 18

— in abdominal parietes, 21

— of gall-bladder, 531, 537

— — liver, 147. See *Pyæmic and Tropical*

— — — diagnosis from hydatid, 59, 171

— — — subdivision into pyæmic and tropical, 165, 166

Active ascites, 450, 480

Acute yellow atrophy of liver, 224

— — — — case of, 236

— — — — its causes, 231, 390, 395

— — — — clinical characters of, 225

— — — — pathology of, 396

— — — — rarity of, 224

— — — — treatment of, 234

Addison's disease, distinction of bronzing from jaundice, 282

— — symptoms of, not due to destruction of supra-renal capsules, 206

Age, its influence on size of liver, 10

ASC

Ague. See *Intermittent Fever*

Alcoholism, a cause of cirrhosis, 239, 269

— — — fatty liver, 48

Amyloid liver. See *Waxy*

Aneurism, abdominal, a cause of jaundice, 344

— of aorta, diagnosis from hydatid, 60

— of hepatic artery, 345

— of sup. mesenteric artery, 346

Animal poisons, a cause of jaundice, 390

Antimony, a cause of jaundice, 395

Appendix vermiformis, gall-stones in, 512

Ascaris lumbricoides. See *Round Worms*

Ascites, active, 450

— causes of, 445

— diagnosis of, from distended urinary bladder, 442

— — — hydatid, 70, 110, 439

— — — ovarian cyst, 434

— — — pregnant uterus, 444

— — — renal cyst, 441

— displacing liver, 18, 217

— from acute peritonitis, 445, 471

— — cancer of liver, 191, 198, 200, 459

— — cancer of peritoneum, 446, 475

— — chronic peritonitis, 445, 473

— — chronic atrophy of liver, 246, 261, 271, 459, 484

ASC

- Ascites from cirrhosis, 246, 261, 459, 484
 — — colloid disease of peritoneum, 448
 — — diseases of chest, 274, 453, 486
 — — — liver, 246, 454
 — — — kidneys, 450, 481
 — — peri-hepatitis, 460
 — — simple dropsy, 449
 — — thrombosis of portal vein, 461
 — — tubercle, 445, 477
 — in diagnosis of cause of jaundice, 429
 — signs of, 432
 — treatment of, 256, 464
 Atrophy of liver. See *Contractions, Simple Atrophy, Acute Yellow Atrophy, Chronic Atrophy*

- B**ATH of nitro-muriatic acid, 128
 Bile, excessive secretion of, a cause of jaundice, 313, 403
 — in blood, not a poison, 297
 — inspissated in ducts, 318
 Bile-acids, formed in liver, 303
 — in urine, a test of cause of jaundice, 422
 — their composition, 303
 Bile-ducts, gall-stones in, 142, 319, 501, 502, 503
 — hydatids in, 67, 90, 94, 97
 — obstruction of, its effects, 142
 — sacculus of, 504, 527
 — tumours in, 337, 367
 — ulcers in, 151, 158, 334
 Bile-pigment, always absorbed, 307
 — in inflammatory exudations, 288
 — — milk, 287
 — — mucus, 287
 — — urine, 286
 — not preformed in blood, 303
 — test for, 283
 Biliary colic from gall-stones, 319
 — — — hydatids, 94
 — — fistulæ, external, 520, 526
 — — internal, 513, 526
 Bilious headaches, 402
 Bitter taste in jaundice, 288
 Bladder urinary, distension of, mistaken for hydatid or ascites, 442, 468

CHR

- Blood-poisons, a cause of acute atrophy, 233
 — — — congestion of liver, 124
 — — — jaundice, 376
 Boils in jaundice, 291
 Bones, diseased, a cause of waxy disease, 29, 31, 35
 Brain, hydatid in, 71
 Bronzing from Addison's disease, 282
 — — sun, 283

- C**ANCER, a cause of fatty liver, 49
 — — — waxy disease, 30
 — colour of skin in, 281
 — of gall-bladder, 540, 543
 — — liver, 187, 543
 — — — case of, 196, 543
 — — — cause of ascites, 191, 198, 200, 459
 — — — — jaundice, 340
 — — — clinical characters of, 187
 — — — diagnosis from hydatid, 61
 — — — — waxy disease, 193
 — — — melanotic, 208
 — — — simulated by inflammation of bile-ducts, 133
 — — — treatment of, 193
 — — — without symptoms, 188, 206
 — — peritoneum, 446, 475
 — — rectum, 543
 — — stomach, a cause of pyæmic abscess, 157
 Carbuncles in jaundice, 291
 Catarrh of bile-ducts. See *Inflammation of Biliary Passages*
 Cerebral symptoms in acute atrophy, 239, 296, 316, 430
 — — — — cause of, 296, 313
 — — — — treatment of, 235, 407
 Chest-diseases, their influence on liver, 13, 123
 Chloroform, a cause of jaundice, 395
 Chlorosis, distinction from jaundice, 280
 Cholestearæmia, 297
 Chronic atrophy of liver, 239
 — — — — a cause of ascites, 246, 459
 — — — — cases of, 260, 484

CHR

- Chronic atrophy of liver from hyperæmia, 240, 251
 — — — — — peri-hepatitis, 241, 252, 273
 — — — — — spirit-drinking, 239, 243. See *Cirrhosis*
 — — — treatment of, 253
 — — — varieties of, 239
 Cirrhosis of liver, 239
 — — — cases of, 260, 268, 484
 — — — clinical characters of, 243
 — — — pathology of, 239
 — — — spurious, 240, 252, 271
 — — — treatment of, 253
 Colic, biliary, 319
 — intestinal, 494
 — renal, 494
 Colloid disease of peritoneum, 448
 Colon, fistulæ with gall-bladder, 516, 534, 543
 Congenital deficiency of bile-ducts, 329
 — malformations of liver, 9
 Congestion of liver, 119, 167
 — — — cases of, 128
 — — — clinical characters of, 120, 167
 — — — causes of, 123
 — — — treatment of, 125
 — — — varieties of, 123
 Constipation, a cause of jaundice, 133, 404, 405
 Contractions of liver, 215. See *Acute, Chronic, and Simple Atrophy*
 — — — spurious, 215
 Copper, a cause of jaundice, 395
 Cystic duct, calculi in, 321, 501, 531

- DEBILITY in jaundice, 295
 Diabetes a cause of atrophy of liver, 53
 — — — — — hypertrophy of liver, 53
 Digestion, derangement of, in jaundice, 289
 Distoma hepaticum, 326, 354
 Dropsy of gall-bladder, 538
 — — — peritoneum, 449. See *Ascites*
 Duodenum, cancer of, 337
 — fistulæ of, with gall-bladder, 514, 529, 534

FIS

- Duodenum, inflammation of, spreading to bile-ducts, 132
 — ulcer of, a cause of jaundice, 332
 — — — symptoms of, 333, 365
 Dysentery, a cause of fatty liver, 49
 — — — — — tropical abscess, 163, 166, 180
 — — — — — waxy disease, 30
 Dyspepsia, simulating disease of liver, 493

- ECHINOCOCCUS. See *Hydatid*, and *Tenia Echinococcus*
 Emphysema of lungs, effects of, on liver, 14
 Endocarditis ulcerative, a cause of pyæmia, 151
 Enlargements of gall-bladder, 142, 145, 213, 536
 — — — liver, 3, 22
 — — — — — rare forms of, 211
 — — — — — spurious, 9
 — — — — — subdivision of, 22
 — — — — — with vitiligoidea, 213, 291
 Enteric fever, a cause of jaundice, 386, 413
 Epidemic jaundice, 133, 388
 Eruptions in jaundice, 291
 Ether, a cause of jaundice, 395

- FÆCES, accumulation of in colon simulating enlargement of liver, 19
 — — — — — cause of jaundice, 347. See *Constipation*
 Fat in fæces, 289
 Fatty heart, its symptoms, 47
 — liver, 43
 — — — case of, 52
 — — — causes of, 47
 — — — clinical characters of, 44
 — — — treatment of, 50
 — — — kidneys, symptoms of, 47
 Feigned jaundice, 284
 Fibroid nodules in liver, 241, 473
 Fistulæ from gall-stones, 513, 527, 529, 531, 534
 — — — — — treatment of, 526

FIS

- Fistulæ from gall-bladder or bile-ducts into colon, 516, 534, 543
 — — — into small intestine, 514, 529, 534
 — — — — pleura, 520
 — — — — portal vein, 519
 — — — — stomach, 513
 — — — — urinary passages, 518
 — — — through abdominal parietes, 520, 531, 534
 — — hydatids, 65
 Fluke of liver, 326
 Frerichs, theory of jaundice, 306
 Fungus hæmatodes of liver, 204

GALL-BLADDER, abscess of, 531

- cancer of, 540, 543
 — carbonate of lime in, 277
 — distension of, from obstruction of duct, 142, 145, 536
 — dropsy of, 538
 — enlargements of, 142, 145, 213, 536
 — — — treatment of, 541
 — — — diagnosis from abscess of liver, 538
 — — — — — hydatid, 60, 539
 — inflammation of, 505, 527
 — — — treatment of, 525
 — perforation of, 505, 527, 529, 531
 Gall-stones, cases of, 145, 360, 527
 — cause of fistulæ, 513, 526
 — — — inflammation of biliary passages, 134, 137, 328
 — — — peritonitis, 505, 527
 — — — pyæmic abscesses, 151, 158
 — how to find them in fæces, 324
 — in appendix vermiformis, 512
 — common duct, 142, 318, 504
 — — cystic duct, 501
 — — duodenum, 511
 — — gall-bladder, 499, 424, 539
 — — hepatic duct, 503
 — — ileum, 509, 525
 — — radicles of hepatic duct, 502
 — — sacculus of common duct, 504, 527
 — — large voided per anum, 507

HYD

- Gall-stones obstructing bile-ducts, 142, 318, 501, 504
 — pathological consequences of, 499
 — symptoms of, 319
 — treatment of, 349, 524
 — vomited, 508
 Gangrene of liver, 100
 Gin-drinker's liver, 239. See *Cirrhosis*
 Gout, a cause of inflammation of bile-ducts, 133, 136
 Glycocholic acid, 303

HÆMORRHAGES from portal obstruction, 248, 266, 267, 463

- in acute atrophy, 231
 — — jaundice, 294, 331
 — into peritoneum in cancer of liver, 202, 204
 Headaches, bilious, 402
 Heart, hydatids in, 71
 Heart-disease, cause of ascites, 453, 486
 — — — atrophy of liver, 240, 251, 274
 — — — congestion of liver, 123, 128
 — — — pyæmic abscesses, 151
 Hepatic artery, aneurism of, 345
 — its function, 308
 — neuralgia, 495, 497
 — pain. See *Pain in Liver*
 Hepatitis. See *Abscess* and *Peri-hepatitis*
 Hydatid of liver, 54
 — — — bursting through abdom. parietes, 65
 — — — — into bile-ducts, 67, 90, 94, 97, 324, 325, 354
 — — — — lungs, 63, 106
 — — — — pericardium, 64
 — — — — peritoneum, 64
 — — — — pleura, 63, 104, 105
 — — — — stomach or intestines 66
 — — — — vena cava inferior, 69
 — — — cases of, 83

HYD

- Hydatid of liver, cause of marasmus, 70
 — — — — — pressure, 70
 — — — — — secondary hydatids, 70
 — — — — — clinical characters of, 54
 — — — — — diagnosis from abscess of liver, 59, 171
 — — — — — aneurism, 60
 — — — — — ascites, 439
 — — — — — cancer, 61
 — — — — — enlarged gall-bladder, 60, 539
 — — — — — pleuritic effusion, 60
 — — — — — renal cyst, 114
 — — — — — fluid of, 61
 — — — — — gangrene of, 70
 — — — — — inutility of medicines in, 73
 — — — — — modes of termination of, 62
 — — — — — operation by small puncture and closure of opening, 75, 83, 85, 89
 — — — — — large permanent opening, 81, 90
 — — — — — prevention of, 72
 — — — — — spontaneous cure of, 62, 112
 — — — — — statistics of operation in, 112, 118
 — — — — — suppuration of, 70, 90, 97, 99
 — — — — — treatment of, 72
 Hydatids compressing spinal cord, 71
 — — — — — ureter, 71, 107, 109
 — — — — — in brain, 71
 — — — — — heart, 71
 — — — — — lungs, 63, 71, 106
 — — — — — multilocular, 212
 Hydrops cystidis felleæ, 538
 Hypertrophy of liver. See *Enlargements*
 — simple, 52

ICTERUS. See *Jaundice*

- Icterus neonatorum, 282, 323, 401, 431
 Ileus from gall-stones, 509, 525, 529
 Inflammation of biliary passages, 131, 328
 — — — — — cases of, 135

JAU

- Inflammation of biliary passages, causes of, 132, 328
 — — — — — clinical characters of, 132
 — — — — — treatment of, 134
 — of gall-bladder, 505, 531, 537
 Innervation, disordered, a cause of jaundice, 398
 Intercostal neuralgia, diagnosis from hepatic pain, 492
 Intermittent fevers, a cause of jaundice, 379
 — — — — — waxy liver, 30
 Intestinal colic, 494
 Intestines, fistulæ into gall-bladder, 514
 — hydatids in, 66
 — ulceration of from gall-stones, 512
 — ulcers of, a cause of pyæmic abscesses, 150
 Itchiness in jaundice, 290
 — — — — — treatment of, 358

JAUNDICE, causes of, 312, 316

- causes of obstruction of bile-ducts, leading to, 316
 — causes of, where no obstruction, 312, 317, 375
 — concomitant symptoms of, 284
 — definition of, 279
 — derivation of, 279
 — diagnosis of causes of, 422
 — diagnosis from spurious jaundice, 280
 — feigned, 284
 — from accumulation of fæces in colon, 347
 — — acute atrophy, 395
 — — aneurism, 344
 — — animal poisons, 390
 — — antimony, 395
 — — cancer of liver, 190, 196, 200, 340, 341
 — — chloroform, 395
 — — congenital absence of ducts, 329, 363
 — — congestion of liver, 122, 403, 421
 — — constipation, 313, 404
 — — copper, 395

JAU

- Jaundice from defective oxygenation, 312, 400, 407
- — distomata, 326
 - — enlarged glands in portal fissure, 340
 - — epidemic causes, 388, 396
 - — enteric fever, 386, 413
 - — ether, 395
 - — excessive secretion of bile, 313, 403
 - — foreign bodies in ducts, 327
 - — gall-stones, 318, 360, 471, 504
 - — hydatids, 67, 90, 94, 324
 - — inflammation of duodenum, 328
 - — — — bile-ducts, 132, 328
 - — malarious fevers, 379
 - — mercury, 395
 - — mineral poisons, 398
 - — nervous causes, 312, 398
 - — obstruction of bile-ducts, 316, 318
 - — — — cases of, 360
 - — — — treatment of, 348
 - — peri-hepatitis, 331
 - — phosphorus, 393
 - — pneumonia, 401, 420
 - — poisons in blood, 312, 376
 - — pressure on ducts, 339, 355
 - — pyæmia, 390, 418
 - — relapsing fever, 381
 - — scarlatina, 387, 415
 - — snake-bites, 392
 - — spasm of ducts, 336
 - — stricture of ducts, 329, 331
 - — tumours of bile-ducts, 337, 355, 367
 - — — — kidneys, 343
 - — — — liver, 340
 - — — — omentum, 343
 - — — — ovaries, 348
 - — — — pancreas, 342, 369
 - — — — stomach, 341
 - — — — uterus, 348
 - — ulcers of bile-ducts, 334
 - — — — duodenum, 332, 365
 - — typhus, 383, 408
 - — yellow fever, 378
 - importance of recognizing causes of, 279

LIV

- Jaundice in new-born children, 282, 323, 401, 431
- in pregnancy, 348, 431
 - independent of obstruction of bile-ducts, 317, 375
 - — — — cases of, 408
 - — — — treatment of, 405
 - intermittent, 428
 - localities of, 284
 - secretions in, 286
 - spurious, 280
 - tabular view of causes of, 316, 317
 - theory of, 300
 - — — in cases of obstruction, 300
 - — — where no obstruction, 302, 312
 - two forms of, 300
 - varying intensity of, 285
 - with vitiligoidea, 291

- KIDNEY**, diseases of, causing dropsy, 450, 481
- — — which do not cause dropsy, 452
 - large cyst of, simulating hydatid of liver or ascites, 115, 441
 - tumour of, causing jaundice, 343
 - — — displacing liver, 19, 115
- Kidneys in waxy disease, 28

- LACING**. See *Tight-lacing*
- Lardaceous liver. See *Waxy Liver*
- Lead-colic, 494
- dusky colour of skin from, 281
- Leucine in acute atrophy of liver, 229
- in typhus, 384, 408
 - its composition, 230
 - and tyrosine, their indications, 299, 377
- Leukæmia, a cause of hypertrophy of liver, 53
- with atrophy of liver, 275
- Lichen in jaundice, 291
- Liver, area of dulness of, 5, 8
- boundaries of, 3
 - contractions of, 215
 - dimensions of, 3

LIV

- Liver, enlargements of, 3, 22
- situation of, 3
- spurious enlargements of, 9
- symptoms of its diseases, 2
- Lungs, disease of, a cause of congestion of liver, 123
- — — — — dropsy, 453
- — — — — jaundice, 401
- Lungs, hydatids in, 63, 71, 106
- Lymphatic enlargement of liver, 212

- M**ALARIA, a cause of acute atrophy of liver, 233, 390
- — — — congestion of liver, 124
- — — — jaundice, 379, 390
- — — — tropical abscess, 166
- dusky colour of skin from, 281
- Malarious fevers, a cause of jaundice, 379
- Malformations of liver, 9
- Marasmus from hydatids of liver, 70
- Melanotic cancer of liver, 208
- Mercury, a cause of jaundice, 395
- its action in diseases of the liver, 126, 309, 404
- Mesenteric artery, aneurism of, 346
- Milk in jaundice, 287
- Mineral poisons, a cause of jaundice, 393
- Multilocular hydatid of liver, 212

- N**ERVOUS influences, a cause of acute atrophy of liver, 232
- — — — jaundice, 312, 398
- Neuralgia, intercostal, 492
- of liver, 495, 497
- Nitro-muriatic acid bath, 128

- O**BSTRUCTION of bile-duct, 142, 318, 501
- — — a cause of enlargement of liver, 142, 536
- — — — — jaundice, 143, 318, 504
- — — cases of, 145, 359
- — — causes of, 316
- — — clinical characters of, 142, 318, 501

PER

- Obstruction of bile-duct, treatment of, 349, 356
- of portal vein, its symptoms, 244
- Œsophagus, cancer of, 222
- Omental tumour, a cause of jaundice, 342
- Ovarian cyst, diagnosis of, from ascites, 434
- — opening into rectum, 466
- Ovarian tumour, a cause of jaundice, 348
- — displacing liver, 18
- Oxygen, deficiency of, a cause of jaundice, 312, 400, 407
- P**AIN in liver, 491
- — — causes of, 495
- — — diagnosis of, from gastric dyspepsia, 493
- — — — — intercostal neuralgia, 492
- — — — — intestinal colic, 494
- — — — — pleurisy, 493
- — — — — pleurodynia, 491
- — — — — renal colic, 494
- — — varieties of, 495
- — — with jaundice, 23, 428
- Painful enlargements of liver, 23, 119
- Painless enlargements of liver, 23
- Pancreas, tumour of, a cause of jaundice, 342, 367, 369
- Paracentesis in abscess of liver, 178
- — ascites, rules for, 257
- — — successful, 275
- — — hydatid of liver, 75
- Paraplegia from hydatid tumour, 71, 111
- Penis, melanotic cancer of, 208
- Perforation of bowels from gall-stones, 512
- — gall-bladder, 505, 527, 529, 531, 534
- Pericardium, effusion in, its effect on liver, 14
- hydatid of liver, opening into, 64
- Peri-hepatitis, a cause of ascites, 460
- — — atrophy of the liver, 241, 252, 273
- — — — obstruction of bile-duct, 331

PER

- Peri-hepatitis, causes of, 241
 — result of syphilis, 241, 330
 — symptoms of, 252, 497
 Peritoneum, cancer of, 446
 — colloid disease of, 448
 — dropsy of, 449
 — fluid in, 432. See *Ascites*
 — — encysted between diaphragm and liver, 18, 205
 — gas in, 218
 — hæmorrhage into, 202, 204
 — hydatids in, 64, 110
 Peritonitis, acute, 445, 471
 — cancerous, 446
 — chronic, 445, 473
 — from gall-stones, 159, 505, 527
 — tubercular, 445, 477
 Phosphorus, a cause of jaundice, 393
 Phthisis. See *Tubercle*
 Pleura, effusion in, its diagnosis from hydatid, 60
 — — — effect on liver, 14
 — fistula of into bile-duct, 529
 — hydatid of liver, opening into, 63, 104, 105
 Pleurisy, pain of simulating that of peri-hepatitis, 493
 Pneumonia, a cause of jaundice, 401, 420
 — its effect on liver, 14, 401
 Pneumo-thorax, its effect on liver, 14
 Portal obstruction, symptoms of, 244, 462
 — vein, compression of, 462, 488
 — — diseases of, 461
 — — fistulæ of into bile-ducts, 519
 — — its function, 308
 — — plugging of, 461
 — — pressure on, by hydatid tumour of liver, 71, 111
 Pregnancy, a cause of acute atrophy of liver, 232, 389
 — — — jaundice, 232, 348, 431
 Pregnant uterus, diagnosis from ascites, 444
 Pulmonary emphysema, its effect on liver, 14
 Pulse in jaundice, 293, 430
 Pyæmia, a cause of jaundice, 390, 418

SPA

- Pyæmia, from hydatids of liver, 70, 99, 100, 151
 Pyæmic abscesses of liver, 147
 — — — — cases of, 154
 — — — — causes of, 150
 — — — — clinical characters of, 147
 — — — — in the tropics, 166
 — — — — treatment of, 152
 Pyelitis, a cause of pyæmic abscesses of liver, 151
 — from hydatid tumours, 71, 107, 109
 Pythogenic fever, a cause of jaundice, 386, 413

RECTI muscles, rigid contraction of simulating enlargement of liver, 20

- Red atrophy of liver, 242, 253
 Relapsing fever, a cause of jaundice, 381
 Remittent fevers, a cause of jaundice, 379
 Renal colic, diagnosis of from hepatic pain, 494
 Rheumatism, its complication with jaundice, 291, 421
 Rickets, influence of on area of dullness of liver, 11
 Right axillary line, 7
 — dorsal line, 7
 — mammary line, 7
 Round worms in bile-ducts, 327
 — — a nucleus of gall-stones, 327

SACCULUS of bile-duct containing gall-stones, 504, 527

- Scarlatina, a cause of jaundice, 387, 415
 Simple atrophy of liver, 219
 — — — case of, 222
 — — — causes of, 220
 — — — its clinical characters, 220
 Simple hypertrophy of liver, 52
 — induration of liver, 241, 252
 Snake-bites a cause of jaundice, 392
 Spasmodic stricture of bile-duct, 336

SPI

- Spinal cord, pressure on by hydatid, 71, 111
 Spleen, enlargement of from portal obstruction, 246
 — — — — waxy disease of, 27
 Spurious cirrhosis, 240, 252, 271
 — enlargements of liver, 9
 — jaundice, 280
 — pains in liver, 491
 Stomach, catarrh of, a cause of inflammation of bile-ducts, 132
 — fistula of into gall-bladder, 513
 — hydatid of liver opening into, 67
 — tumours of, a cause of jaundice, 341
 — ulcers of, a cause of peri-hepatitis, 241, 253, 273
 — ulcers of, a cause of pyæmic abscesses of liver, 150, 155
 Stools in jaundice, their importance, 289, 426
 Stricture of bile-ducts, 329
 Suppression of bile, 302
 — — — — arguments against it, 303
 Suppuration of gall-bladder, 537
 Supra-renal capsules, cancer of, 206
 Sweat in jaundice, 287
 Syphilis, a cause of acute atrophy of liver, 232
 — — — — congenital absence of bile-ducts, 330
 — — — — peri-hepatitis, 241, 330
 — — — — waxy liver, 30, 37, 41

TÆNIA echinococcus, 61, 72

- Taste, bitter in jaundice, 288
 Taurocholic acid, 303
 Temperature in jaundice, 293
 Thrombosis of portal vein, 461
 Tight-lacing, its effect on liver, 11
 Tropical abscess of liver, 162
 — — — — cases of, 180
 — — — — causes of, 166
 — — — — clinical characters of, 167
 — — — — diagnosis of, from abscess of gall-bladder, 538
 — — — — — — — enlarged gall-bladder, 172
 — — — — — — — hydatid, 171

TYR

- Tropical abscess of liver, diagnosis of, from pyæmic abscesses, 172
 — — — — may burst in different directions, 174, 175, 183
 — — — — objections to pyæmic theory of, 163
 — — — — pathology of, 162
 — — — — puncture of, 174, 186
 — — — — — — — arguments for, 177
 — — — — — — — objections to, 176
 — — — — — — — rules for, 179
 — — — — — — — treatment of, 172
 Tubercle, a cause of fatty liver, 30, 48
 — — — — waxy liver, 30
 — coexistence of, with cancer, 222
 — of liver, 139, 211
 Tubercular enlargement of liver, 139, 211
 — peritonitis, 445, 477
 — vomicae, a cause of pyæmic abscesses of liver, 151
 Tumours between diaphragm and liver, 18
 — intra-thoracic, effect of on liver, 14
 — compressing bile-ducts, 142
 — in bile-ducts, 337
 — of kidney, a cause of jaundice, 343
 — — — — displacing liver, 19, 115
 — of omentum, a cause of jaundice, 343
 — — — — displacing liver, 19
 — of ovary a cause of jaundice, 348
 — — — — displacing liver, 18
 — of pancreas, a cause of jaundice, 342, 367, 369
 — — stomach, a cause of jaundice, 341
 — — uterus, a cause of jaundice, 348
 — — — — displacing liver, 18
 Tympanitis excessive, simulating atrophy of liver, 216
 Typhoid state, pathology of, 228, 296, 380
 Typhus, a cause of acute atrophy of liver, 233
 — — — — jaundice, 383, 408
 Tyrosine in acute atrophy of liver, 229

TYR

- Tyrosine in typhus, 384, 408, 410
 — its composition, 230
 — and leucine, their indication, 299, 377

ULCERS of bile-ducts and gall-bladder, 151, 158, 334

- — — a cause of pyæmic abscesses, 150, 155, 505
 — — — — — peritonitis, 505, 527
 — — — — — stricture, 335
 — — bowels from gall-stones, 512
 — — duodenum. See *Duodenum*
 — — stomach, a cause of fatty liver, 49
 — — — — — peri-hepatitis, 241, 253, 273
 — — — — — pyæmic abscesses of liver, 150, 155

Uræmia from hydatid tumour, 109

Ureter, compressed by hydatid tumour, 71, 107, 109

Urinary passages, fistulæ of with gall-bladder, 518

Urine in acute atrophy of liver, 229, 236

- — — nephritis, 451
 — — contracted kidneys, 452
 — — fatty kidneys, 47, 451
 — — jaundice, 286, 422
 — — waxy disease, 28, 452

YEL

Urine, test for bile-acids in, 423

— — — bile-pigment in, 283

Urticaria in jaundice, 291, 421

Uterus. See *Tumours*

VAGINA, fistula of with gall-bladder, 519

Vena cava inferior, hydatid opening into, 60

— — — pressure on, by hydatid tumour, 70

— portæ. See *Portal Vein*

Vertebrae, cancer of, 206

Vitiligoidea, 213, 291

WAXY liver, 23

— — cases of, 35

— — causes of, 29

— — clinical characters of, 24

— — diagnosis of from cancer, 193

— — nodulated variety of, 26, 42, 193

— — prevention of, 31

— — treatment of, 33

XANTHOPSY in jaundice, 295

YELLOW atrophy of liver. See *Acute Atrophy*

— Fever, 378

— vision in jaundice, 295, 372



NATIONAL LIBRARY OF MEDICINE



NLM 01182301 3